THE

TREATMENT OF NERVOUS DISEASE

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THE TREATMENT

OF

NERVOUS DISEASE

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TO

SIR ARTHUR MITCHELL

K.C.B., M.D., LL.D.

WISE IN COUNSEL

FIRM AND STEADFAST IN FRIENDSHIP

THIS BOOK IS DEDICATED

PREFACE

The framework round which the following chapters are built consists of certain lectures delivered from time to time in the Royal Infirmary. A few of these appeared in imperfect and immature form in the *Scottish Medical and Surgical Journal.* These have been carefully revised and largely added to, and the information they contain has been amplified and brought up to date. It has seemed wise to adopt the somewhat colloquial and pliant diction of spoken lectures, as this permits of a certain elasticity and flexibility of style otherwise impossible.

It is, however, to be understood that but a small part of that which appears in the following pages has been actually delivered in lecture form. By far the greater portion is new, and is now published for the first time.

The idea which underlies the structure and arrangement of these lectures is that the physiological function of the particular part of the nervous system which in each case happens to be affected determines the variety of treatment required. Consequently, in the case of all the lesions of any particular set of neurones identical or analogous in function, there is much in common as regards the therapeutic measures which ought to be adopted. It will be found, for example, that as regards the various lesions which may affect the lower motor neurone and the several diseases hereby produced, the lines of treatment are fundamentally and in essence the same, though in detail they may and do differ according to the precise nature of the morbid process and the exact point of its attack.

Following this physiological arrangement, it has been found possible to group the various nervous diseases, as regards treatment, under the heading of that set of neuronic fibres which, in each instance, is chiefly and mainly involved. This arrangement is, of course, in no sense a perfectly accurate one, for many diseases involve

various sets and varieties of neurones, differing in function. But it has been found to be a convenient method, seeing that it enables the whole subject to be treated within moderate compass. For it is clear that if the treatment of one or two typical diseases in any group is fully described, it is not necessary to discuss with any minuteness the details of management of other and perhaps very rare disorders affecting the same neurones, the treatment of which must necessarily be conducted on similar lines.

This book is not, therefore, in any sense an exhaustive work. It is intended, rather, to be suggestive and to stimulate independent thought.

J. J. G. B.

3 Chester Street,
Edinburgh, *December* 1904.

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LECTURE I

ON PROPHYLACTIC MEASURES

Introductory remarks—Prophylaxis in diseases of the nervous system — Etiology of these diseases — Heredity — The characteristics of neurotic children—Their training and management—School life—Mental work—Prophylaxis in neurotic adults.

THERE is no system in the body the diseases of which present more discordant results as regards treatment than do those of the nervous system. Some respond readily to the means you employ, and complete recovery results. Others run a slow or a rapid course, apparently quite uninfluenced as to ultimate result by any sort of treatment you may choose to adopt. Even in the most hopeless cases, however, it is usually in our power to afford more or less relief, while in the more favourable the application of remedial measures is sometimes brilliantly successful. *Introductory remarks.*

Believing that the treatment of disease of the nervous system is too often conducted on mere

routine principles, that measures are taken without much consideration of the definite objects they are meant to serve, or the ends they are capable of attaining, I wish in these lectures to put before you some kind of general survey, however imperfect in detail, of the methods at your command for the management and treatment of such cases, and of the indications which direct us in the use of these methods.

General results of treatment. We should not approach such a subject with too sanguine views, for, except in the case of a few diseases, the results of treatment are attained slowly and not without much painstaking endeavour. Nor, on the other hand, should too pessimistic an attitude be assumed, for this may end in disheartening the patient and may send him to the nearest quack. It is only by endeavouring to grasp the principles which underlie nerve treatment, so far as they are known, and the indications for and limits of usefulness of those remedies the action of which we only know empirically, that we can treat wisely such diseases of the nervous system as may fall under our care. The quack is ever with us, though he is by no means poor; and if we do not master the methods of employing such a remedy

as electricity, for example, patients, much to their subsequent sorrow, tend to drift towards unqualified electricians, rubbers, and the like, who profess to make such methods a speciality, and who surround them with a seductive mystery. No one of the methods about to be described is beyond the use of the intelligent practitioner, nor is there one of them which he ought not to be able to employ from time to time as need arises.

Question of prophylaxis.

Before we proceed to consider the lines of treatment which may be adopted in cases of such diseases, there arises the problem of prophylaxis —of the ways in which nervous disease may be warded off; a subject not, as a rule, very fully discussed in medical works.

Importance of details.

Prophylaxis, so far as it is successful, owes that success to attention to a vast number of details, of points in management, which individually indeed may appear trivial, but the sum of which is of a high importance. As it is of consequence that you should consider and understand these points, I shall speak of them in some detail, and this the more, seeing that great success may attend well-directed efforts

towards preventing the establishment of nervous disorders.

Causes of nervous disease.

All diseases of the nervous system depend for their establishment in the body upon some proximate or exciting cause, the result of environment, and almost all owe their origin also to certain remote causes which are known to us in connection with heredity.

Proximate causes.

Considering first and very briefly the exciting or proximate causes, it is clear that many are beyond our control. We cannot, for example, prevent the occurrence of traumatism, nor can we arrange so that undue exposure to cold and wet shall certainly never take place. The movements of a floating kidney can, however, be controlled, and I have frequently seen an associated neurasthenia considerably improved thereby.

Fevers, including syphilis and tubercle, the toxines of which are so apt to attack the nervous tissues, have a prophylaxis of their own which we need not consider here; nor shall we deal with the methods of guarding against the occurrence of cerebral hæmorrhage and thrombosis—methods which are fully discussed in works on general medicine. It is the part of the surgeon to see that a cerebral abscess does not follow an

otitis, that infection of the mastoid does not occur, and that wounds of the scalp are kept aseptic. It is not necessary here to refer to the means to be used to prevent poisoning by lead in those exposed to the action of the salts of that metal, nor to the necessity of careful moderation and regulation in the use of alcohol, if indeed it be used at all. Excesses *in venere* as well as *in vino* are important, fortunately also avoidable, causes of nerve lesion; and intellectual work, when it is combined with worry and anxiety, breaks down many men.

Peripheral irritation.

Sometimes, on the other hand, the immediate cause may be found in some peripheral irritation. Adenoids or other nasal or tonsillar condition, if present, should be got rid of, as should also any decayed teeth which may be present. Parasitic worms in the intestinal tract are credited with the production of infantile convulsions in some cases, and I know of one instance, at least, in which mania followed the invasion of the œsophagus by a round worm.

Disease of reproductive organs.

Peripheral irritation produced by disease of the reproductive organs is a fruitful source of nervous disorder, but very great care should be exercised in dealing with such conditions.

Particularly should you be cautious of allowing a neurotic woman, especially if young and unmarried, to pass under any prolonged gynecological treatment. So far as my experience goes, such treatment in these cases is more often productive of harm than good. I should like you to lay down this general rule for yourselves—namely, that unless there be some very definite uterine lesion which can unquestionably be removed within a short time, no gynecological treatment should be adopted in neurotic women. There are, of course, exceptions to this rule, and each case should be judged on its own merits; but without any doubt, prolonged treatment which is always directing the patient's attention anew to these organs, and which involves repeated vaginal examination, often does harm in neurotic women. And much the same rule applies in the case of neurotic men. Any local urethral treatment (save for gonorrhœa or other definite local malady) should only be allowed, if allowed at all, after grave consideration.

Care needed in dealing with these.

A full consideration of prophylaxis should include within its scope all of these and many other similar causes. Such as are more closely connected with the nervous system will be referred

to again. The remainder are rather to be looked at from the point of view of general medicine, and these need not detain us here.

Predisposing causes.

Even of more importance than these proximate causes is the predisposition to nervous affection; those causes which, acting by heredity, render the tissues of the nervous system more easily injured by any unfavourable condition of environment which may occur.

Heredity.

It is well known to you that many nervous diseases are in their origin traceable to the influence of heredity. I do not now refer to that singular group of family diseases of which Friedreich's Ataxia may be taken as a type. These are directly developmental in origin, and it is doubtful whether any post-natal prophylaxis would influence them. I refer rather to those inherited nervous disorders which appear from time to time in neurotic families; such as epilepsy, asthma, hysteria, neurasthenia, insanity, neuralgia, megrim, exophthalmic goitre, alcoholism, morphinism.

The neurotic tendency, the nervous diathesis, on which such diseases take root, is very widely spread; and in families so affected individual

members here and there fall vietims to its miseries, but fortunately not all. For before these maladies ean arise not only must there be an inherited tendeney, but there must also be an exeiting eause at work.

A neurotic child.

By a nervous or neurotie ehild I mean one who has inherited a delieate, mobile, and unstable nervous system, eapable of reaeting in speeial ways to special stimuli. He is the outeome of civilisation, of a highly specialised method of living. You must remember that the laws of the struggle for life and the survival of the fittest apply with but little force to eivilised man. The whole tendency of our laws, of our publie health arrangements, of medical science, is to prevent the weak man from going to the wall. In the words of one of the most distinguished thinkers of this seat of learning,[1] addressing our profession, "we recognise that in the weakest there is a potentiality of strength, in the worst there is a potentiality of good—and it is our business to see that nothing is lost that we ean help to save." One of my friends, desiring to give form to this view of eivilisation, has had depieted on his book-plate a strong man,

[1] Professor Crum Brown, in a Graduation Address (1889).

blind, who carries on his shoulders a cripple, acute of vision. Together these two, combining their faculties, make their way through life. Such then is civilisation, and under its sway weak men are protected and survive, the nervous system tends to develop to an abnormal sensitiveness, and hence come our neurotic children.

Now, in persons hereditarily liable to nerve disease, much can be done by way of warding off the incidence of such maladies, by strengthening the body, toning up the nervous system, and training the mind to preserve an equable balance. To reach its best results this method of prophylaxis must be commenced in the young. We may therefore now consider what are the indications in a child which suggest this nervous inheritance, which point towards the probability of future nerve disease, and which should, therefore, excite our apprehension.

Indications of neurotic inheritance.

Parents

And of these come, in the first place, the deductions to be drawn from observation of the parents. If one parent is distinctly neurotic, the child will probably be so likewise, and this probability is greatly enhanced if both parents show indications of the nervous diathesis.

In the second place, most valuable deductions

Appearance of child. may be drawn from the general appearance of the child. The neurotic child is usually rather stunted in growth, but is wiry and muscularly strong, so far at least as quick movements are concerned. His features are small, fine and delicate, his expression alert, his manner restless, his disposition impulsive and wayward, and at the same time he is usually very clever at lessons. Emotional and excitable, he is ready to blush on the slightest occasion. Of such is the "infant prodigy," the joy and pride of a doting and neurotic mother.

But while he is as a rule clever, he is not capable of making any continuous and persevering effort. This is often seen in connection with the play of such a child. For example, he may be seized with a violent desire to make some toy. He procures the materials and eagerly sets about its construction. But, long before completion, he tires of the idea and casts it aside for some fresh and different pursuit.

Marks of degeneracy. Then you will sometimes find more distinct signs of degeneracy, indicating at once the history of the past and the dangers of the future. You may notice, for example, that the development of body or of mind is retarded,

that there is marked asymmetry of the head or face, or that the ear is abnormal in shape or in size. You may detect the presence of hare-lip, or of cleft-palate, or of that high arching of the palate with which the researches of Dr. Clouston have made us familiar. The lower jaw is often developed out of proportion to the other bones of the face, giving the heavy jaw of the neurotic, frequently seen in criminals. On the other hand, in some neurotics the chin is ill developed, small and retreating. The conditions of spina bifida and of club-foot are also very suggestive.

Changes in behaviour.

There are, moreover, sundry other points in the behaviour and condition of the child which are fitted to cause us anxiety. The lack of self-control, a passionate temper, the want of the joyous mirth proper to childhood, and of the admirable and kitten-like foolishness of that age, the presence of a seriousness and thoughtfulness unnatural in a child, or of a curious mental lethargy, all point to a neurotic inheritance. The like may be said of that solitariness which we sometimes see in such children, leading them to dislike companions and even their near relatives, of strange sexual perversions and inversions which may occur, of that melancholy

by which they are sometimes seized, and whieh not uneommonly brings thoughts of suieide —thoughts sometimes even translated into aetion.

Suicidal tendency.

There are, indeed, few forms of insanity so markedly hereditary as the suieidal tendeney. Hammond (*Treatise on Insanity in its Medical Relations*: London, 1883, p. 79) narrates a ease of this kind. A gentleman, well-to-do in the world, cut his throat in a warm bath at the age of thirty-five. He left two sons and one daughter. The eldest son eut his throat at thirty-five. The seeond committed suieide in the same way at about the same age. The daughter eut her throat at thirty-four. Her son tried to eommit suieide in the same way in a warm bath at the age of twenty-seven. He again made an unsuceessful attempt in this fashion at the age of thirty, and in the following year he sueeeeded in committing suicide, using preeisely the same method as his grandfather had employed. In this very striking example of hereditary suicidal insanity there was, no doubt, a strong imitative faetor.

Religious peculiarities.

Often untruthful, suspieious and quarrelsome, untidy and even dirty in their habits, neurotic

children sometimes exhibit curious phases of a sentimental, unreal and emotional religion. I recollect the case of a boy of thirteen or fourteen, brought under my notice some years ago, who arranged with himself that when he heard the name of God, even in ordinary conversation, he would lay himself prostrate on the ground. He did this whether he were in the house or out of doors. If I remember rightly, he used this Great Name himself, on occasion, if he thought that his friends abstained from giving him sufficient opportunity for the display of this form of religion. We decided that the best way to treat the condition was to pay no manner of attention to his peculiarities, and in a short while this curious religious phase passed away.

Moral obliquities at time of puberty.

It is important to note that at the time of puberty, both in boys and in girls, certain well-marked moral obliquities are not uncommon. These are, as a rule, of short duration, ceasing when the worst of the strain, which the onset of puberty occasions, has passed off. But unless their true nature is realised, much injustice and harm may be done. For example, a child, formerly truthful, may begin to tell lies. These are often objectless and usually stupidly absurd.

Or a child may suddenly take to stealing, the symptom developing in some cases into regular kleptomania. This form of vice—if in the circumstances you can call it such—is a further development of the curious craze for collecting useless odds and ends which some neurotic children show. This again is probably related to the abnormal eagerness many adults shew to make collections. This collecting mania appears to be very contagious. An epidemic of the kind, consisting in the collection of picture post-cards, is at present sweeping over Germany and even threatening our sane shores!

Other signs of neurotic inheritance. You will also attach importance to the occurrence of megrim, night terrors, somnambulism, convulsions in teething, stammering and backwardness in learning to speak. Even bad spelling is, I take it, a sign of neurotic inheritance. Astigmatism and colour-blindness occur frequently in neurotics, as do also such symptoms as incontinence of urine, cyclic albuminuria and the various tics. It need hardly be added that the occurrence of any distinct nervous disease in a child is very significant.

A child exhibiting marks of neurotic inherit-

ance may, if his education and management be judiciously conducted, develop into an adult strong in body and in mind. Indeed, we owe to neurotics much that is highest in science and in art, the strongest and most polished literature —that touch of genius which stirs the world. A useful and even a distinguished life may therefore be his lot. But on the other hand he may sink, and his years may be passed in a miserable melancholia or other form of insanity. If, therefore, a physician be consulted on the upbringing of such a child, he ought to remember how great a power for good or for ill is exerted by careful environment and by sagacious education. To be successful in this, great attention to detail is required, and no point should be regarded as trivial which has any bearing on the matter in hand.

Good effects of careful management.

In the first place, if the mother be a neurotic and hysterical woman, it is advisable that the child should not be much with her; and this because her irritable temper and her unbalanced mind render her unsuitable to exercise due management and control, and again, because any nervous manifestations in the mother are apt to

To whom should the child be entrusted?

be copied and adopted by the imitative child. If the mother is at all sensible she will herself recognise the wisdom of this. But it is often difficult to arrange for such separation (even if it be only for some hours a day), because such a mother possesses an almost exaggerated affection for the neurotic child who so clings to her. A sensible, calm and judicious governess or children's nurse should, if possible, be entrusted with the management of the child.

Regulation of life.

As to the kind of life which such a child should lead. It should be one of regular orderliness, a life of air and of sunshine (both literally and metaphorically), of breezy common sense in which cold bathing plays its part in toning up the nervous system, and in which muscular exercise is strongly insisted on. By all means advise gymnastics if you like, but games which interest are, I think, better, and a good tumbling romp is the natural and healthy outlet for the high spirits of a child. As he gets older he should be encouraged to swim (particularly in the sea), to ride, to cycle, to golf, to play hockey, tennis, etc. Children's parties are not for him, nor theatres, nor evening concerts, but he should be encouraged to form friend-

ships with other children and to meet them in games.

Exercise. Although I think games a better form of exercise for neurotic children than school gymnastics, yet from one point of view the practising of gymnastic feats is certainly good, viz. that such attempts tend to overcome that abnormal fear of hurting themselves, which such children feel. They may also help in keeping within bounds the feeling of giddiness which is apt to trouble nervous persons in looking downwards from some height.

Vertigo. This giddiness, which many Alpine climbers know, reaches a high degree in neurotic persons, and every effort ought to be made to overcome it. Most persons can do so by practice. It is a sensation which is painful, which may sometimes be dangerous, and which deprives the subject of it of much pleasure in life.

It is highly important, as regards the child's mind, that everything which is subjective should be discouraged and suppressed, and everything which is objective encouraged. His interest in plants, in natural history, particularly in looking after animal pets, in machinery, in gardening, and the like, should be stimulated. Those

artificial parties at which children are taught and expected to imitate the manners of their elders, are very objectionable. They take away that freshness and childishness which it ought to be one of our objects to preserve as long as possible. A child should be kept childlike. It is robbery to take his freshness from him, and it is worse than folly to teach him to think and act and feel as an adult.

Food. His food ought to be simple, and the cooking good but plain. The object being to build up the body—bone, muscle and fat—and not to stimulate the cortex, you will see that the child is fed chiefly on milk, eggs, cream, porridge and other farinaceous food, poultry, fish and rabbit; and that butcher meat is only given occasionally. Such is the Gospel of Fat which my friend Dr. Clouston so successfully preaches.[1] It need hardly be added that alcohol should not be given in any form to young neurotics; indeed, its use even in adults of this temperament is dangerous.

Sleep. The necessity for a full measure of sleep is great in all children, but specially so in those who

[1] You may bear in mind that all healthy children, and young women up to the age of twenty, are, or ought to be, plump.

are neurotic. A child of twelve requires at least ten hours of sleep, and sleep is so precious a gift that nothing should be allowed to interfere with its full measure. Sleeplessness means either that the child is ill, or that his brain is being overworked.

It is very important that the love of nature should be encouraged. The love of flowers and their cultivation attract some, that of animal pets others, and it is always possible to cultivate, in some direction, that interest which the naturalist feels in things round him. *Love of nature.*

A cold bath in the morning is useful in giving tone to the nervous system. It accustoms the skin to rapid change of temperature, and thus obviates the risk of chills. The importance of this is greater in the case of neurotic children than in others, because in them the circulation in the skin is under an abnormally mobile nerve control. This is shown by the fact that such children blush with extraordinary ease, and that they are subject to more variation in the temperature of the body than normal children are. For such reasons the practice of douching the skin with cold water is specially useful in the case of neurotic children. *Cold baths.*

The child must be very delicate who can be harmed by allowing the contents of a jug of cold water to fall on his head and shoulders, while he stands with his feet in warm water.

Behaviour of those in charge.

The behaviour of those in charge of such a child is a very important matter. They should be calm, judicious, firm, and yet kindly. There must be no show of temper or irritability, even when the behaviour of the child is most trying.

Imitativeness.

All children are imitative, but neurotic children are specially so, and they imitate with a peculiar fidelity what may be wrong in the deportment or doings of those around them. It is hardly necessary to say how harmful it is to the child to witness signs of friction between the parents.

If the parents have any reason to be anxious as to the state of health of the child, all signs of such anxiety should be hidden from him, for a neurotic is only too apt to attach importance to his symptoms and to analyse them. Such thoughts tend to hypochondriasis.

The management of such a child is not to be in any way hard or rigorous, nor one depending on threats, yet it is exceedingly important that

implicit and unquestioning obedience should be exacted on all occasions. So far as possible, when a rule displeasing to the child has to be laid down, the reason for the measure should be explained to him. Above all things, never tell a child anything which is untrue, even with a view to his own good.

A neurotic child is very apt to imagine unfairness, to brood in silence, it may be for years, over a supposed injustice. He is very sensitive in his feelings, easily inflated by injudicious praise, easily depressed by indiscreet blame.

Rules of management.

The mistake which parents most commonly make in regard to neurotic children is to spoil them by indulging every wish; yet the opposite of this is quite as harmful. A neurotic child surrounded by a net-work of iron rules will emerge with his spirit crushed and broken. Rather should he be allowed to carry out his own wishes (provided these are not distinctly harmful), even if he suffer discomfort by so doing. Let him learn by the hard but wholesome way of experience. Better far that he should so suffer than that he should be brought up like a marionette, to move in response to every wire

the parent touches. It is important to train the child to act on his own judgment and to think for himself, so that when he becomes a man he may not suffer from that weakness and fickleness of purpose so often the lot of the neurotic.

Restraining passions. An equal balance of mind is not easily attained, but this should be the great object in view in the rearing of such children. It should be constantly inculcated how necessary it is to restrain the passions of anger, jealousy and the like, to combat the temptations of the palate, and, if necessary, other less innocent appetites, *Pain.* to face pain bravely and in silence. For pain is a great educator, and neurotic children, who probably feel pain more acutely than others, ought to be taught to control themselves, and on ordinary occasions at least not to give expression to the pain they may feel. The firm attempt to limit and repress the feeling reduces the amount of suffering experienced.

The fear of hurting themselves is very prominent in the minds of neurotics. Active games and exercises, which involve some little risk of hurt and pain, are therefore good educators.

All the senses indeed are very acute in

Acuteness of sensation.

neurotics. The sense of taste leads them to form strong likes and dislikes to special articles of diet. Such peculiarities should be repressed as far as is reasonable. The sense of hearing is also hypersensitive. Such children cannot bear loud noises, which indeed pain them, and prefer to sit in a quiet room. This hypersensitiveness to sound can be overcome by practice, and this is one reason why school-life is so good for neurotic children.

Hypersensitiveness to the opinions of others as to his own appearance, bearing, or conduct, is a frequent source of suffering to a neurotic child—a suffering which will continue through life unless it is repressed. This can only be done by impressing on him how very unimportant a person he is, and that so long as he acts rightly, it does not matter what others think.

Punishment.

As to punishment, when that is needed, I hold strongly that it should hardly ever take the form of corporal chastisement, and certainly never in the case of highly neurotic children. I have known severe and lifelong damage result from the mental impression produced by such treatment. It is easy enough to make so susceptible a nature as that of the neurotic child feel the

displeasure his conduct has aroused and the disappointment his parent or teacher feels. The touch of personal vanity in the neurotic child makes such knowledge of itself a punishment.

Sentimentality and fancy.

All signs of sentimentality in such a child should be suppressed by those in charge, and they should attach, or rather appear to attach, as little importance as possible to any complaints of illness he may make. Little should be made of such complaints, and it is almost superfluous to say that the details of the illnesses of other persons should not be discussed in the hearing of a neurotic child.[1] And as the whole strength of the teacher should be directed to develop the *mens sana in corpore sano*, therefore that which pertains to fancy and sensuous enjoyment should be discouraged, for in this lies much danger of relaxation of mental fibre. A dreamy idealism, an artistic bent, exciting and sensuous music, everything which stimulates the imagination and partakes of mysticism ought to be avoided.[2] A perfervid, sentimental and hysterical religion

[1] In the case of girls at first menstruation, care should be taken to explain quietly that the process is a natural one.

[2] In what has been said I do not wish to indicate that the higher imaginative faculties should not be cultivated. In the Bible, Milton, Dante, the student finds, in lofty imagination, the highest ethics and the highest wisdom. These ennoble and strengthen man.

does much harm, and sometimes awakens feelings which are far from being religious. A more true and useful Art, if such be latent in the child, may safely be encouraged later, when body and brain are more fully developed. At the same time the child should not be brought up in too matter-of-fact a way. He should be taught to understand and to enjoy wit, and what measure of the saving grace of humour he may have should be encouraged in every possible way. To be able to see the humorous side of an otherwise grave matter may, in future, tide him over serious anxieties.

Books read. Too great care cannot be exercised in selecting such books for a neurotic youth as are healthy and manly. Lifelong damage may be done by a single perusal of an objectionable or impure book. Careful guard has to be kept against sexual aberrations. This implies some oversight as to the companionships he may form, and so brings us to the question of the choice of a school.

School life. The advantages which school life offers are found both in day-schools and in boarding-schools, though some are more prominent in the

latter. They consist in the sense of an organised discipline such as is impossible at home, in the feeling that no exceptions can be made without serious cause, but that every child must conform to rule. This is an important matter for neurotic children, and not less so is the tendency of school life to rub off the angles and to tone down the nervous peculiarities of such children, to encourage a spirit of manliness and *esprit de corps.* I have repeatedly seen boarding-school life prove most effective in these ways in the case of neurotic children. On the other hand, boarding-schools have their peculiar dangers, and much depends on the wisdom, common sense and acuteness of the master in charge of the house in which the boys sleep.

Choice of school. For all but the most profoundly neurotic children school is advisable. Whether the child should be sent to a boarding-school or to a day-school depends in part on whether the boy can stand a rough-and-tumble life, and in part on whether he is under wise control at home. But, whatever the school, it should be borne in mind that a wise superintendence and the presence of a manly and healthy moral tone are of far

greater importance for the future of the boy than mere teaching power.

School work. As to school work, you will find that neurotic boys usually take a high place. They do this either because they learn so easily that mental work is light to them, or because they are over-conscientious in their labours. In the former case no great evil may be done, but in the latter the mental life and vigour of the future may be seriously endangered. For such a boy the stimulus of competition is bad, and he may leave school at once covered with honours and exhausted in brain; with a cerebral cortex so enfeebled that no good, solid work can be done by him in after life. Every one must have noticed instances of this kind, not only in school but also in University life. But if a boy is good at games as well as at lessons, you need not fear for him, though he come out top of his class or figure as Ettles' Prizeman or Senior Wrangler.

It is of course important to encourage the love of work. Whether that work be mechanical, in the manufacture of a toy, for example, or whether it be mental, the sense of satisfaction in work well done is a good tonic for the nervous system. In youth, particularly at the time of

puberty, the whole waking hours should be occupied, whether in work or play. Any want of occupation is bad, encouraging idle and wandering thoughts, which sometimes tend to become vicious.

Over-work. There is a curious difference between childhood and adult life as regards the effect of mental work and of worry on the brain. In the adult it is difficult to injure the brain by over-work, because, when a certain intensity of strain has been reached, the brain refuses to work any longer and the man is forced to rest. On the other hand it is quite easy to do serious injury to the brain of a child by pressing him with mental work, and this happens even more readily in neurasthenics than in healthy children. The reverse is the case with regard to worry. This falls lightly on a healthy child, and the most serious trouble may at any time be dissipated by the administration of a sugar-plum. In a man, on the other hand, cares and worries are not readily shaken off. The mind cannot easily forget them, and sleep is interfered with. A neurotic child takes an intermediate position in this matter. In him worries and anxieties are apt to fix themselves in a quite morbid fashion.

Mental exhaustion.

In conclusion a word may be said as to mental work and mental exhaustion in neurotic children. Even in the perfectly healthy child we know from many observations that an hour or two of such work produces an exhaustion which is quite perceptible, as it blunts the tactile sense to a degree which can be measured. In neurotics exhaustion of the cortex is even more readily produced, the visual field, for example, becoming distinctly smaller. This points to the necessity of making the periods of work of a neurotic youth short, and of interposing muscular exercise between each. It might be possible in an individual case, by direct testing of the visual or tactile sense, to say whether too much exhaustion was being occasioned, or, in other words, whether the mental work done was proving too much for the boy.

Effect of mental work on perception of skin sensation.

Griesbach[1] made a careful study of the effects of school work on the power of perception of cutaneous sensation. He employed Weber's method of two points, estimating the distance which these points had to be separated in order

[1] Griesbach, "Ueber Beziehungen zwischen geistiger Ermüdung und Empfindungsvermögen der Haut," *Arch. f. Hyg.* Bd. 24 (1895), S. 124. The table from which the figures given above are taken will be found on p. 175 of that article.

that when they touched the skin they should be pereeived as two distinct points. According to his observations (and they have since been confirmed by other experimenters) the more severe the mental work the more the threshold rises, and the greater must the distance be between the points,—in other words, the more is perception blunted. This was specially the case where the work done involved writing. After cessation of the sehool work it required two hours of rest before sensation had regained its normal acuteness. Let me give one example, where sensation was measured on the ball of the thumb. The school work began with an hour of mathematics. Before its commencement the threshold was 6 m.m., at its termination it had risen to 10. The next hour was devoted to Latin and the following one to Greek, at the end of which the threshold had risen to 13.5. During the subsequent hour the lesson was one on religion, which must have so far relieved the mental strain, for perception became more acute and the threshold fell to 9. The study of elementary physics in the next hour raised the threshold again to 11 m.m. After these labours it required two hours before normal perception was regained and

Example of blunting of sensation by mental work.

the threshold had fallen to its original figure. There seems no reason why an intelligent teacher should not apply this test to ascertain whether a nervous child is being over-pressed at school.

Prophylaxis in adults.

For an adult who comes of a neurotic stock the same general line of life should be adopted. It should be carefully explained to him or to her, as the case may be, that whatsoever makes for bodily hardness and strength is to be aimed at. Mental strain should be avoided, brain work being done rather in the early hours of the day than late at night. A due amount of exercise is to be taken in the open air. But, unfortunately, persons of this cast of mind do not always take the proper kind of exercise. How often, on urging upon such a man the necessity of exercise, he replies, rather indignantly perhaps, that he walks regularly so many miles a day. And how often have we to reply, "That is not exercise." For a neurotic man thinks while he walks; he turns problems over and over in his mind, arranges the outline of his next sermon, sketches a lecture, or devises some mercantile scheme. The mind therefore gets no rest. What is urgently required is that the exercise such a man

Exercise.

takes should be interesting to him and should be absorbing. He cannot think as he rides a fresh horse, as he cycles, as he plays golf. Pursuits of this kind are the exercises for a neurotic man or woman, and mere walking,—the ordinary "constitutional,"—is not exercise at all in the proper sense.

Dangers of alcohol.

Allusion has already been made to the dangers of alcohol in such persons, and these dangers are great. It is in neurotics that we see that form of alcoholism which consists in outbursts of hard drinking with intervals of sobriety. You should make it a rule never to recommend the use of alcohol in any form to a neurotic without grave consideration. And the same remark applies to morphia with perhaps even more force. If you do see good reason to give that or other similar drug, arrange so that its administration is not in the hands of the patient nor the dose at his discretion.

And of narcotics.

Cocaine is more dangerous still. It should never be administered to a neurotic for any condition which would require its frequent repetition. I have known cocaine, used as a nasal spray, produce an almost irresistible craving in a neurotic person.

In conclusion, may I venture to hope that in

this brief and necessarily imperfect sketch of the principles which should regulate the management of neurotics to the end that actual nervous disease may be warded off, you may find some thoughts which may be of service to you also in guiding such patients towards a healthy robustness of mind and of body?

Note.—For those readers who are specially interested in the question of the education of neurotic children, a copious and growing literature exists. The publications of the various associations for child study, at home and in America, contain much valuable material. Among the special works on the subject mention may be made of the following: Warner, *The Nervous System of the Child*, London (1900); Kirkpatrick, *Fundamentals of Child Study*, New York (1903); Oppenheim, *Nervenleiden und Erziehung*, Berlin (1899); Heller, *Grundriss der Heilpädagogik*, Leipzig (1904). To these volumes, and particularly to the two last named, I am indebted for certain valuable suggestions made use of in the preceding pages.

LECTURE II

ON TOXIC NERVOUS DISORDERS AND THEIR TREATMENT

Toxic nervous diseases—Definition of term "toxine," and of term "neurone"—Varieties of toxic agents—Morphological changes in toxic conditions—Selective action of toxines—Treatment of toxic nervous disease—Withdrawal of poison—Elimination—Antagonism by drugs and by anti-toxines—Theory of anti-toxine action—Tetanus and its anti-toxine—Botulism—Morphia and the morphia habit—Alcoholism—Syphilis as affecting the nervous system—Details of anti-syphilitic treatment—Para-syphilitic maladies.

Toxines as affecting nervous tissues.

In the last lecture I discussed the various lines which prophylaxis should take in those who are hereditarily predisposed to nervous disease, explaining in some detail the points which, I think, should be kept in view in the upbringing of neurotic children. To-day I propose to speak of a problem of great importance, and one which meets us at every turn when we are considering the treatment of nervous disease—the subject of the toxines which affect the nervous tissues.

Definition of the term toxine.

Were we to regard the subject from a purely pathological point of view, we should have to define very carefully what we mean by a toxine. There are those who restrict the term to such poisons, the result of bacterial action, as possess at least two specific atom groups, a haptophorous and a toxophorous, and which, when administered to animals, give rise to the production of a corresponding anti-toxine. We shall have to consider this aspect of the subject presently and in some detail. But for the moment you may note that, so far as the diseases of the nervous system are concerned, we should be obliged, were we to adopt so narrow a view, to restrict the term toxine to the poisons of diphtheria, of tetanus, and of botulism. Abrin and ricin no doubt would fall within the definition, but they are outside our present subject.

On the other hand, we know that many nervous lesions are produced by bacterial poisons of the exact nature of which we are uncertain. We have reason to believe that some are true toxines, in Ehrlich's sense; others are probably more of the nature of ptomaines or amides. And in our lack of accurate knowledge regarding this supremely interesting subject, I shall not, I

think, materially err if I use the term Toxine somewhat loosely to cover all those poisons which result from bacterial action.

The term neurone.

The nervous system is made up, as you know, of masses of neurones arranged in sets, and linked up in such a way as to form paths along which nervous stimuli may pass, centripetally or centrifugally, as the case may be. These neurones differ, of course, in function,—some conveying motor, others sensory, or, more correctly, centripetal impressions. These are broad distinctions. But even amongst those which are motor there are fundamental differences in position, relations, structure, and function. These differences and divergences are seen to be more numerous and more marked when we come to consider the various centripetal neurones. Tactual impressions, feelings of heat, of cold, of pain, are in each case conveyed by neurones which are specially differentiated for each individual sensation. To these must be added the numerous neurones conveying the various impressions which we speak of under the term "muscular sense," some of which are perceived, though most are subliminal, never rising to consciousness.

The term "neurone," which will be con-

stantly met in these lectures, is used to designate that physiological entity which is formed of the cell-body, its nucleus, its protoplasmic processes, its axis-cylinder throughout the whole length, and its terminal arboresations. The neurone theory has of late suffered strenuous attack from many sides. It is not the purpose of these pages to discuss the arguments for and against that theory. It need only be said that while in certain particulars and directions the theory as originally stated appears to break down, yet, taking it as a whole, I still believe in its substantial truth. The term "neurone" is a very convenient one, both clinically and pathologically, and may well be adhered to, under the definition just given, without thereby necessarily implying a rigid belief in the original theory of Waldeyer.

The neurone theory.

All these various neurones, differing as they do in position, relations, structure, and function, differ also in their reaction to poisons and toxines. Some are susceptible to one, some to another; and as we proceed to consider the subject in detail you will recognise as the dominant factor the selective action of these poisons. Some, such as lead and tetanus, attack motor neurones. In the case of others only sensory neurones are

Selective action of toxines.

affected. Many poisons, however, attack both centripetal and centrifugal neurones.

Results of toxic action.

The nervous system, as a whole, is very easily affected by the various toxic agencies, and a considerable number of nervous diseases arise from such poisoning. Most forms of peripheral paralysis take origin in this way, as do also in all likelihood many of the so-called systemic diseases of the cord. Many cortical affections, some varieties of insanity, uræmia, diabetic coma, morphinism, the various forms of alcoholism, and probably certain affections commonly looked upon as functional, may be fairly considered as due to poisoning with some one of the many forms of toxine which attack nervous tissues.

It must thus be apparent to you that the subject of toxine poisoning is of a high importance in its bearing on nervous disease, and probably I do not go too far in saying that the future of nerve treatment lies to a large extent in the direction of anti-toxines.

The action of these various poisons on the nervous tissues, although it has attracted great attention of late years, and has formed the subject of much research, is as yet very imperfectly understood. Still less do we understand the

nature of the action of anti-toxines on the elements of the neurone. But enough light has now been thrown on this intricate and obscure subject to show the surpassing interest of the problems involved, and to give some idea of the wideness of the therapeutic field which will be opened to us as our knowledge increases.

The various toxic agents.

Let us then, in order to get some grasp of so complex a subject, consider in the first place what are the toxic agents which may so act on the nervous system as to give rise to the numerous forms of lesion which are traced to such poisoning. Without attempting an exact classification, for which indeed our knowledge does not fully suffice, the following enumeration may give you some idea at once of the extent and of the complexity of the subject.

Among the metals which act as poisons on the nervous tissues may be mentioned lead, mercury, arsenic, antimony, aluminium, phosphorus.

Many organic compounds also attack the neurones. Of these the following may serve as examples: alcohol, ether, chloroform, antipyrine, trional, sulphonal. These act sometimes as acute poisons, but more commonly their action is

exhibited clinically in the chronic form. Morphia, cocaine, strychnine, nicotine, and ergotine may also be added to this group.

The toxines of diphtheria, tetanus, rabies, syphilis, influenza, typhoid and other fevers, of bubonic plague, of malaria, are all special poisons for the nervous tissues, though they differ much in potency, some being powerful to a high degree, while others are not nearly so dangerous. To these must be added the toxines of certain other infective lesions affecting mainly, if not exclusively, the nervous system, such as Landry's paralysis, Beri-Beri, acute anterior polio-myelitis, polio-encephalitis.

Auto-intoxication. Nor must we forget to add to our list those nervous lesions which arise from auto-intoxication—that is, poisoning with the products of a faulty metabolism, or from the retention of products which ought to have been eliminated. Of this class are the phenomena of uræmia, and the nervous lesions which occur in diabetes. Possibly those which show themselves in pernicious anæmia may also belong to this group.

Gastro-intestinal auto-intoxication. Then there is a group of nervous lesions which are occasioned by toxic bodies produced by the decomposition of food and absorbed from the

alimentary canal. The most striking of these is that very fatal affection which we know as botulism, of which I shall say something presently, but there are many lighter manifestations of poisoning of this sort which meet us in practice. The longer the food lies in the stomach the more likely is it to undergo decomposition, and it is, consequently, chiefly in cases of gastric dilatation that such symptoms arise, and this the more in that the deficiency of gastric juice, and particularly of hydrochloric acid, so constantly present in that malady, favours early putrefactive change.

The less severe forms of nervous affection resulting from gastric dilatation take the shape of neurasthenia, and from what I have seen I am convinced that many of the cases of neurasthenia which we encounter owe their origin to auto-intoxication of this kind. But more severe and striking symptoms may follow such poisoning. Tetany, for example, is not so very rare a result. *Tetany.* Moynihan[1] notes five cases of this kind, and Dickson[2] has recently described an exceedingly interesting example of tetany resulting from

[1] Moynihan, "A Note on Gastric Tetany," *Practitioner*, vol. lxx. (1903), p. 354.

[2] Carnegie Dickson, "A Case of Gastric Dilatation and Tetany," *Practitioner*, vol. lxx. (1903), p. 44.

auto-intoxieation. In other cases severe mental symptoms[1] may be produeed by gastro-intestinal poisons, and even aeute hæmorrhagie eneephalitis has been known to result.

From this enumeration, whieh I have not attempted to make exhaustive, you will readily perceive how many and how various are the toxines whieh attaek the nervous struetures.

And now it beeomes neeessary to eonsider what the changes are whieh these toxines are capable of producing.

Action of toxines on the neurone.

While it is hardly possible to doubt that those neurones whieh are affected are attacked as a whole, the protoplasmic proeesses, the cell body, the nucleus, and the axis-cylinder or axone all suffering, though not perhaps equally or in exactly the same way, yet the staining methods at eommand limit our observations, so far as aeute poisoning is concerned, almost entirely to the nerve eell itself. Indeed, it is only reeently that studies of the aetual results of acute toxic poisoning on nerve eells have been rendered

[1] Raimann, "Ein Fall von Cerebropathia psychica toxæmica (Korsakoff), gastro-intestinalen Ursprunges," *Monatsschrift f. Psychiatrie und Neurologie*, Bd. 12 (1902), S. 329.

possible, and this chiefly by the application of the method of Nissl and its modifications.

Our knowledge of the changes to which these toxines give rise is due in great measure to the investigation of the results of experimental poisoning in animals. A considerable amount of work of this kind has been done during recent years by such observers as Nissl, Marinesco, Babes, Hodge, Lugaro, van Gehuchten, Pandi, Berkley, Flatau, Goldscheider, and others.[1]

Pathological changes.

This is not the place nor the time to discuss pathological changes, but it may be said in a word that the microscopical appearance of cells poisoned by these various toxines differs somewhat according to the individual toxine which has acted. In general there occur such changes as chromatolysis, swelling of the cell and of the nuclear bodies, and various forms of degeneration. Take, for example, poisoning with tetanus toxine. In this case the large motor cells of the ventral horns become swollen, the nuclei become large and pale, the Nissl's bodies increase in size and break up in granular disintegration. It may

[1] The paucity in the amount of the experimental toxine work which has been done in this country is due to the operation of the Anti-vivisection Acts, the administration of which has so crippled British, and especially Scottish, workers in this and other similar fields of inquiry.

interest you to note that the morphological changes in these cells caused by strychnine closely resemble those of tetanus,—interesting, that is, in view of the similarity in the symptoms of the two conditions. I must, however, add that certain recent observations[1] appear to show that these cell changes are the result of extreme fatigue rather than of direct toxic action. *Strychnine.* If a frog, to which a very large dose of strychnine has been given, be placed in water at freezing temperature, no such cell changes are observed.

The cell changes which follow poisoning with various toxines (metallic poisons, organic compounds, the toxines of disease) are the result of a chemical process which probably depends on the presence in the nerve cell of certain atom-groups which have an affinity for atom-groups in the toxine.

These alterations are the more pronounced and the more rapid in their development in direct proportion to the degree of concentration of the poison. But you should carefully remember that, though they are expressive of a corresponding change in function, there is no

[1] Gordon Holmes, "On Morphological Changes in Exhausted Ganglion Cells," *Zeitschr. f. allgem. Physiologie*, Bd. 2 (1903), S. 502.

direct or traceable proportion between the amount of microscopic change and the intensity of the nervous phenomena thereby occasioned. Probably indeed other changes take place in the cells which the staining process to which they are subjected does not reveal, for, in the case of tetanus toxine, for example, the clinical phenomena may increase at a time when the morphological changes, having reached a height, are showing signs of restitution. On the other hand the converse may obtain (as in malon-nitril poisoning), the function being apparently completely restored before the microscopic changes have disappeared.

Relation of microscopic change to function.

A word may now be said as to the micro-organisms which give rise to so many of these toxines. They may themselves penetrate the nervous tissues, and Babes has found certain forms within the nerve cells. The bacillus of plague, for example, passes by the small vessels of the grey substance of the cord to invade the nerve cells there. Those micro-organisms which do penetrate the nerve cells do not, however, seem to cause harm so much by their mere presence as by the toxines which they evolve. But most of these organisms do not attack the

Micro-organisms.

nervous tissues direetly, and only produee their effeets there by means of the toxines whieh they manufaeture in other parts of the body.

Selective action of toxines.

Before we leave this part of the subjeet there are three points whieh should speeially engage your attention. In the first plaee, you should have elearly in mind that eertain nerve eells show an affinity for eertain toxines, while others do not. The weight of the tetanus toxine, for example, falls on the eells of the ventral horns of the cord. Aleohol again, as Nissl has shown, produees marked changes in the eortieal eells, while those of the neighbouring *cornu ammonis* remain unaffected.

The seeond point, to whieh indeed allusion has already been made, is that in those instanees in which the same nerve eells are affeeted by different poisons the appearanees so produeed vary. Arsenie, lead, stryehnine, for example, all affeet the cells of the anterior horns, but the difference in the mieroscopic changes so produeed is fairly definite

Individual idiosyncrasies.

To these two points, to whieh Nissl and other observers attach great importanee, I should like to add a third, namely, that the same toxine

may act differently and on different neurones according to the individual it is attacking. Alcohol does not act in the same way on every man who drinks more than is good for him. His speech bewrayeth one man, inco-ordination of gait another, disturbance of mental functions a third. Lead poisoning in one may produce the symptoms of multiple sclerosis (as in the case I showed you a few weeks ago), in another those of locomotor ataxia, in a third those of the ordinary drop-wrist. Nor does the toxine of influenza always attack the nerve cells, nor, if it does so, is the neurone affected always the same. These differences, which are very important clinically, probably depend on some inherited or acquired weakness or peculiarity of certain neurones, rendering them more than usually susceptible to the attack of the toxine.

Treatment of toxic nervous conditions.

And now, when you come to consider what is to be done by way of treatment in those various toxic forms of nervous disease, one point at once becomes clear. In cases in which the poisoning is still going on you must, if possible, put a stop to that at once. If the drinking water contains lead, that source of danger must be cut off. The

man who is being poisoned with alcohol must be made to abstain, the patient suffering from malarial neuritis should be moved to a healthy locality, and the best results in the case of Beri-Beri are obtained when the infected district is forsaken. The production of poisons of bacterial origin in the gastro-intestinal tract may be limited, to some extent, by the administration of drugs which affect the vitality of the micro-organisms. This involves the question of gastro-intestinal antiseptics, which we shall consider in a future lecture.

Elimination of toxines.

These are sufficiently obvious points, and it is equally clear that we should use every means in our power to encourage the elimination of the toxine from the system. In the case of lead, for example, we do this by the administration of iodide of potassium which, forming a soluble iodide of lead, allows of the ready excretion of the poison by the intestines, kidneys, and skin, while, in order that no re-absorption from the bowel may occur, we see that the patient takes sulphate of magnesia at suitable intervals, so that the lead in the intestinal tract may be converted into the insoluble sulphate. When we are dealing with the toxines of fever some good may be

attained by stimulating excretion through the skin. In the case of myelitis following an infective fever, for example, profuse diaphoresis induced by a vapour bath may do good, and this form of treatment is specially important in the early stages of multiple neuritis, and in those cases of neuritis which are of rheumatic origin.

Lavage of the stomach.

Reference has already been made to the fact that the changes which occur in the contents of a dilated stomach are apt to give rise to toxines which may occasion more or less serious symptoms. In such cases it is clear that the proper procedure is to remove these contents by means of careful and thorough lavage. The washing-out should be performed regularly, and in some cases even twice a day. If this, combined with the use of suitable intestinal antiseptics, fails, recourse may be had to the operation of gastro-enterostomy.

Lavage of the tissues.

A more generally efficient method of removing toxines from the body consists in flooding the tissues with fluid. This can be done to a certain extent by means of subcutaneous or intravenous injections of saline solutions. These processes are, however, painful, not very efficacious, and, in some cases, dangerous. A much more satis-

factory method is that by means of rectal injections of large quantities of saline solution. The procedure recommended by Wernitz[1] is that which I have employed, and it is probably the most convenient.

Method of procedure. A rectal tube of medium length is introduced into the rectum and passed up as far as possible without occasioning pain. It is connected with an irrigator filled with warm 1 per cent saline solution. This vessel is placed about a foot above the level of the pelvis of the patient, and the solution allowed to run very slowly into the bowel. The speed should be such that it takes about an hour for a litre of fluid to pass. During this time the bowel occasionally contracts and, when this occurs, the irrigator should be lowered and the fæculent fluid allowed to pass back. This is then replaced by fresh fluid and the injection continued. After the first injection of about one litre, by which the bowel is largely cleared out, a pause of one or two hours should be allowed. Then another injection of one litre is made with equal slowness. These operations should be repeated at intervals throughout the

[1] Wernitz, "Zur Behandlung akuter Infektionskrankheiten," *Therapeutische Monatshefte*, 1903, S. 2.

day and night. In this way very large quantities of fluid are absorbed into the body and the tissues are washed in saline solution. The result on the temperature, in cases of pyrexia, is very favourable, and much fluid escapes by the skin and by the kidneys, the urine increasing rapidly until it sometimes measures five or six litres in the twenty-four hours. There can be no doubt that toxines are readily got rid of in this way, and in some cases the bacteria themselves may be thus swept out.

Antagonising of toxines.

But you approach a more interesting part of the inquiry when you come to consider the employment of those drugs which have what may be called a specific action in antagonising certain toxines.

If there were any doubt in your minds as to the possibility of the occurrence of this action, such dubiety would be set at rest most completely by such investigations as show that at the same time as the clinical toxic symptoms are repressed by the remedy, the pathological appearances are equally and simultaneously held under control. One of the best examples of this kind is furnished by the phenomena of poisoning with malon-nitril,

and the control which hyposulphite of sodium exerts on that process.

Poisoning with malon-nitril.

In 1896 Heymans and Masoin showed that by means of hyposulphite of sodium the results of poisoning with malon-nitril ($CN - CH_2 - CN$) and other allied nitrils could be successfully combated. More recently the pathological side of the question has been investigated by Goldscheider and Flatau, who give their results in a work on the subject published in 1898. Malon-nitril, when administered to animals, occasions pronounced nervous symptoms, convulsions, vaso-motor disturbances, paralysis and death; but even when the paralysis is extreme and death at hand, rapid recovery follows the administration of hyposulphite of sodium. This rapid restitution of function may be traced morphologically in corresponding changes in the cells of the anterior horns of the cord. When these cells are stained, in cases of malon-nitril poisoning in animals, it is seen that the Nissl's bodies have lost their regular arrangement and contour and are disintegrating, and that the protoplasm lying between has become abnormally and deeply stained. If, however, hyposulphite of sodium be used as an antidote, recovery at once takes place as regards

symptoms, and the pathological changes in the cells are found likewise to disappear, though more gradually; morphological restitution having been observed to be complete in seventy-one hours.

This is perhaps as clear a demonstration as you can expect to obtain of the remedial action of drugs in connection with toxic nervous lesions. Others of a similar kind, though less fully worked out, will no doubt occur to you in connection with your pharmacological and therapeutic studies.

But it is not merely in the case of those toxines which are formed in the laboratory that the action on the neurone can thus be antagonised. For, although the pathological demonstration is lacking, we know from clinical experience that the toxines of certain diseases may be controlled in a similar fashion. In rheumatic neuritis, for example, the salicyl compounds act in this way; in malarial neuritis, quinine; in syphilitic affections, mercury; syphilis is, as you know, a very frequent and serious cause of nervous disease, and the proper carrying out of anti-syphilitic treatment is correspondingly important. To this subject, however, we shall return presently.

Control by drugs.

Anti-toxines. The toxines of such diseases as affect the nervous system may thus be eombated to some extent by drugs prepared in the ehemieal laboratory. They may also be eontrolled by substanees formed in the laboratory of the body. An animal whieh has been rendered immune to an infective proeess has elaborated within its body, and furnishes in its blood-serum, an anti-toxine whieh is eurative against that infeetion. The subjeet of serum therapeuties and protective inoculation is at present limited, so far as nervous affeetions are coneerned, to rabies, to diphtheria, and to tetanus, though in the future it is likely to be eonsiderably extended. I do not suppose that any one now doubts the utility of anti-rabie inoeulation—Pasteur's great work; nor can there be any question as to the diminution in the death-rate of diphtheria sinee the serum treatment has come to our aid. The former, under present eonditions, ean only be earried out in sueh institutions as that of Pasteur in Paris; the latter belongs rather to general medieine than to the therapeuties of nervous disease.

Toxines and anti-toxines. The subject of the aetion of toxines and anti-toxines is of sueh inereasing importanee, and is, as has been already said, likely to enter so

largely in the future into the treatment of nervous disease, that I may perhaps, though very briefly, deal with it here.

Ehrlich's investigations.

Ehrlich's great work commenced by his showing that the phenomena of natural immunity depended on the formation of anti-toxines in the blood of animals to which toxine had been given. He went on to prove that definite quantities of toxine could be neutralised *in vitro* by definite quantities of anti-toxine. This action depends, as he inferred, on some definite relation of a specific atom-group in the toxine to an atom-group of the anti-toxine, so that the one fits the other as a key does a lock. Without going into further detail, it may be said that the toxine molecule consists of two atom-complexes. The one of these, the haptophorous group, combines either with the corresponding group in the cell —the receptor—or with the anti-toxine. The other is the toxophorous group, to which the toxic action is due. You should carefully note that the haptophorous group has no evident morbid action on the cell with the receptor of which it combines. It acts as an intermediary and binds the toxophorous group to the cell, thus allowing the toxine to act; for without

the haptophorous group the toxophorous cannot act.

Immunity. According to this theory, the development of immunity in the animal body consists in the formation of receptors in excess. These are thrown off into the blood-stream. When, now, a toxine is administered, these free receptors unite with the haptophorous groups of the toxine, and as the toxine cannot act without its haptophorous group, by the agency of which the toxophorous group is bound to the cell, the latter is uninjured, and immunity is produced.

Tetanus. This is not the time nor the place to deal in any detail with pathological questions, save in so far as such conditions and processes serve to illumine the question of treatment. I may, however, delay for a moment to consider the subject of tetanus and its anti-toxine, partly because the question concerns us here, and partly on account of certain recent work, which throws light on the subject.

The toxine of tetanus. The toxine of tetanus, though an exceedingly powerful poison, acting fatally in animals in minute doses, very easily loses its virulence. By mere lapse of time this takes place. In a

few days, according to Behring and Knorr,[1] its virulence may fall to only about one-hundredth part of the original amount. Sunlight and the oxygen of the air have a similar weakening effect, and many chemicals act also in this direction. The effect of iodine is perhaps the most interesting. A weak solution of that substance rapidly acts so as to prevent the poison from developing its toxic action, but does not affect its power to immunise.[2] This can hardly have other meaning than that the iodine destroys the toxophorous group, leaving the haptophorous unaffected.

Its mode of action.

Tetanus toxine acts almost exclusively on the nervous tissues. The special degree in which the toxine becomes bound to these tissues was first shown by E. O. Shakespeare,[3] who produced tetanus poisoning in animals by the sub-dural introduction of the nervous tissues of an animal poisoned with tetanus. The correctness of this observation has been frequently confirmed since. Indeed, the specific attraction which the nervous tissues present to the tetanus

[1] Behring and Knorr, "Ueber den Immunisierungswerth des Tetanusheilserums," *Zeit. f. Hyg.* Bd. 13 (1893), S. 407.

[2] Oppenheimer, *Toxine and Anti-toxine*, Jena, 1904.

[3] Shakespeare, E. O., *Centralblatt für Bakteriol.* Bd. 2. (1887), S. 541.

toxine must be extraordinarily strong, for it has been found that in the rabbit, after the intravenous injection of a fatal dose, the whole of the toxine has left the blood within one minute.[1] And there is evidence which goes to show that the toxine becomes bound to the nerve-cells and not, to any appreciable extent, to those of other tissues. Wassermann and Takaki,[2] in their experiments, found that an emulsion of fresh brain substance was able to bind a certain though small amount of the toxine, and to that extent to render it inert. This effect was not produced by an emulsion of the tissues of other organs. No doubt the nervous tissues furnish the greater number of those free receptors which give the anti-toxine its virtue. There is, however, some evidence to show that other tissues take part in their manufacture.

Mode of action. Regarding the exact method of action of tetanus toxine there is some recent and interesting evidence. Marie and Morax[3] have shown

1 Decroly and Ronsse, "Pouvoir toxique et anti-toxique du sang," *Arch. Intern. de Pharmacodyn.* 6 (1899), p. 211.

2 Wassermann and Takaki, "Ueber tetanusantitoxische Eigenschaften des Centralnervensystems," *Berliner klin. Woch.* (1898), S. 5.

3 Marie and Morax, "Recherches sur l'absorption de la toxine tétanique," *Annales de l'Instit. Pasteur*, 16 (1902), p. 818.

that the toxine, after subcutaneous injection, was to be found in the nerve-trunks, and Meyer and Ransom[1] have confirmed this, and have extended our knowledge very considerably. Their observations go to show that the gate of entrance of the toxine to the nerve centres is a very special and singular one, namely, through the peripheral terminations of motor fibres in muscle, and thence up the motor axis-cylinder to the anterior horns of the cord. The poison, though present in the blood circulating through the nerve centres, does not under normal conditions act directly on these, but only reaches them by slow centripetal conduction through motor axones. In this way and on this hypothesis you may explain to yourselves the latent period of this toxine. The incubation period of the disease is, to some extent at least, also due to the time required for the conduction of the toxine along the neurones. But while this is what may be called the normal course of events, you may note that if the nerve centres are in any way injured by a local damage, the toxine finds

Path by which tetanus toxine enters the nervous tissues.

[1] Meyer and Ransom, "Researches on Tetanus," *Proceedings of the Royal Society of London*, vol. lxxii. (1903), p. 26. A fuller account of these observations is contained in a paper by the same authors in the *Arch. f. exp. Path.* Bd. 49, S. 369.

entrance at the injured point, and then you do not have the latent period due to axonal conduction of the poison. Many other interesting points, particularly those bearing on the action of the toxine, under certain circumstances, on sensory fibres, are to be found in a recent paper by W. M. Fletcher.[1] I have dealt with these questions of the action of tetanus toxine in some detail, because, as will be seen immediately, they have considerable bearing on the treatment of the disease.

Tetanus anti-toxine.

The changes in the nerve-cells of the ventral horns produced by tetanus toxine have been already described and need not again be detailed. When, now, tetanus anti-toxine is injected in animals it is possible thereby to control not only the symptoms which tetanus toxine has produced, but also the corresponding changes in the nerve-cells of the ventral horns. Goldscheider and Flatau,[2] experimenting on animals, have shown that small doses of the anti-toxine modify the symptoms and at the same time retard the microscopic changes, while with larger doses,

[1] Fletcher, "Tetanus dolorosus, and the relation of tetanus toxine to the sensory nerves and the spinal ganglia," *Brain* (1903), p. 383.

[2] Goldscheider and Flatau, *Normale und pathologische Anatomie der Nervenzellen*, Berlin, 1898.

especially if these are given early, the symptoms may be made to disappear, and in the affected cells there is a rapid restitution to a normal morphology. This result is, however, due, in all likelihood, not to any anti-toxic action on the toxine in the nerve-fibres, but to the binding of the toxine in the blood. Observations go to show that when the toxine has been injected into a motor nerve, no amount of anti-toxine in the blood can prevent an attack. So far as treatment is concerned, it may safely be said that when you give a dose of anti-toxine under the skin in a case of tetanus, you cannot expect that dose to neutralise the toxine which is already bound in the axones of the motor nerves. The anti-toxine will not do that. What it will do, however, is to prevent any more toxine from entering the nervous system. Nor is it, perhaps, too much to expect, though the point has not been absolutely proved, that if the anti-toxine were injected into the substance of the motor nerve which has been infected, the toxine might then be neutralised. In a case in which death threatened and anti-toxine, administered subcutaneously, had been without curative effect, it might, I think, be justifiable

to inject the anti-toxine into the spinal cord itself. Roux and Borrel[1] have, indeed, obtained very encouraging results in animals by the intra-cerebral administration of the anti-toxine.

Its clinical use.

It is in all probability for the reason just given that the use of tetanus anti-toxine in the human subject has not been by any means uniformly successful. Nevertheless I have no hesitation in saying that the moment the diagnosis of tetanus is made, anti-toxine injections should be commenced, without waiting to see the effect of other forms of treatment.

Preparations of tetanus anti-toxine.

There are several forms of tetanus anti-toxine in use—that of the Lister Institute of Preventive Medicine, that of Roux, that of Behring, that of Tizzoni and Cattani, and others. The anti-toxine of Tizzoni, as prepared by Merck, is sold in the dry form and is to be used as follows. The powder is to be rubbed up and dissolved in distilled water in the proportion of 1 in 10. Of this solution a quantity of from 5 to 10 c.c. or more is to be injected hypodermically from a sterilised syringe, this dose being repeated as seems necessary. The anti-toxine of the Lister

[1] Roux and Borrel, "Tétanos cérébral," *Ann. de l'Instit. Pasteur*, 12 (1898), p. 225.

Institute is supplied in a liquid form, the dose being 100 c.c., to be administered in five injections at the same time, under the skin at different situations.

Botulism. The disease known as botulism is in some respects analogous to tetanus. In certain cases of poisoning by means of impure meat, especially in the form of those doubtful sausages to which the Teutonic palate is so partial, there occur, in addition to intense gastro-intestinal symptoms, various severe nervous phenomena—cramps, convulsions, and bulbar symptoms ending in death. From such meat Van Ermengem[1] isolated the Bacillus botulinus, which on cultivation was found to produce a toxine of high virulence, and which in animals gave rise to the symptoms of botulism as seen in man. This toxine acts specifically on nerve-cells. Kempner and Pollack[2] describe the chromatolysis and other destructive changes which it produces in the cells of the ventral horns of the cord, which indeed had already been noted by Marinesco.[3]

[1] Van Ermengem, "Ueber einen neuen anaeroben Bacillus und seine Beziehung zum Botulismus," *Z. f. Hyg.* Bd. 26 (1897), S. 1.

[2] Kempner and Pollack, "Die Wirkung des Botulismustoxins auf die Nervenzellen," *Deutsch. med. Woch.* (1897), S. 505.

[3] Marinesco, "Lésions des centres nerveux produites par la toxine de bac. botulinus," *Soc. Biol.* 48, 31 (1896); *Sem. méd.* (1896), 488.

Its affinity to nervous tissues resembles that of tetanus, for it is found that the substance of the brain and cord is capable of binding firmly a certain amount of the toxine with which it has been rubbed up. This powerful and dangerous toxine forms within the body a corresponding anti-toxine, but apparently this anti-body is likely to be of little use clinically, for it is found that it will antagonise the toxine only if it is given within the first twelve hours. In all likelihood the toxine of botulism becomes so bound to the neuronic elements that the anti-toxine has little effect. In this particular it resembles tetanus.

Before we leave this part of the subject I must mention more fully three toxic agents, which, in respect of treatment, demand your special attention. These are: Morphia, alcohol, and syphilis.

Morphinism. Opium and its alkaloids are taken, by those who are its slaves, in various ways, but that which is the most frequent in this country is the hypodermic injection of morphia. This disastrous and widely-spread habit is more common among neurotics than in persons of normal constitution,

and although the desire for a fresh sensation, in a life of emptiness and luxury, may sometimes be its starting-point, yet the injection of morphia is usually commenced in order to relieve pain, to control chronic diarrhœa or dysentery, or for some similar purpose. Doctors, with their ready access to the drug, pay a heavy tribute to morphinism, as do also druggists and dentists. Of late I have become increasingly impressed with the necessity of exercising care in giving morphia in private practice. In hospital work, for obvious reasons, the dangers are not so great. But if you give a hypodermic injection of morphia to a delicate woman, for example, for the relief of pain, it is very difficult to resist the appeals she will make to you for its repetition should the pain return. Your hand may easily be thus forced. In chronic disorders not immediately threatening life, and in particular in such regularly recurring painful conditions as dysmenorrhœa, you should exercise great caution in giving morphia. In any case you will do well to retain complete control by invariably administering the remedy yourself. Never should the patient be permitted to use or to possess a syringe, nor should hypodermic medication be

Dangers of giving morphia.

entrusted to a nurse. This latter mistake is, I fear, too often made. The use of morphia should be confined, as far as possible, to two classes of cases—first, those patients who are suffering from some acute and short affection, such as pleurisy or renal colic, and, second, those dying of some painful malady.

The habit of injecting morphia, once commenced, can hardly be shaken off by the victim himself. Help is always required. But before speaking of this it may be well that you should have some idea of what the symptoms of morphinism are.

Symptoms of morphinism.

The patient, having used morphia, let us suppose, to check severe pain, is tempted to continue its use after the illness is over. He does this because of the feeling of comfort and wellbeing which each injection induces. Gradually he increases the daily quantity, and presently he finds that he cannot get on without the stimulus of morphia. As the grip of the poison tightens on him the demand of the body for its daily dose becomes more imperious. If the unfortunate morphinist makes a struggle to keep control over the habit, he uses his syringe only at stated times in the day. As the hour

of injection approaches he becomes seized with great restlessness. He cannot attend to work, he cannot think save of the one subject,—morphia; and he feels weak and miserable. He has fits of yawning, crouches over the fire, and shivers with a cold perspiration. The moment the injection is taken he passes into euphoria. He becomes a new man—warm, comfortable, alert, bright, ready for mental or bodily work, although not for work of a high order.

Character of morphinist.

The character of the morphinist very rapidly deteriorates and he becomes an expert and plausible liar. Disorderly in his habits, procrastinating, wayward and capricious, the victim of this craving sinks into an utter selfishness.[1]

This first stage of morphinism, which may be called that of euphoria, lasts for a variable time, perhaps for a year or more, its duration depending partly on the individual but chiefly on the amount of morphia which is taken daily. The smaller the dose the longer will this first stage last.

But presently there comes a time when the use of the syringe no longer gives such satisfaction,

[1] The change in character is, however, not so strongly marked here as it is in alcoholism.

when under imperious demand from the body, morphia is used in increasing doses and with increasing frequency. The tissues of the body then show more evident signs of suffering from the poisoning. Emaciation comes on, the face becomes sallow and haggard, there are signs of gastric catarrh and there is usually obstinate constipation. The mental functions now show evident indications of failure, and memory becomes weak and imperfect. The muscular system also suffers enfeeblement and little exertion produces fatigue. During this stage the injections become painful, induration takes place round each prick of the needle, and frequently abscess formation results. Such patients are sometimes so scarred that there remains hardly an area of the skin of normal appearance.

Struggles against the habit.

It is important that you should remember that in his struggles to regain mastery over himself,—for most morphinists do so struggle at one time or another,—he usually attempts to compass the diminution of morphia by replacing it with some other drug. Thus he comes to add alcoholic indulgence to morphinism, and if he has resort to cocaine, then the last state of that man is infinitely worse than the first.

As to the quantity of morphia which such persons administer to themselves, you will find that there is considerable variation. During the first stage the daily consumption may amount to 3 or 4 grains perhaps. In the later periods 15 to 20 grains is not an unusual daily allowance, and many take quantities greatly in excess of these figures.

Treatment of morphinism.

There is no doubt that the treatment of a confirmed morphinist is best conducted in a hospital or asylum, or in a well-managed nursing home. To attempt it in the house of the patient is to increase enormously the natural difficulties of the situation. But in any case it is worse than useless to try to break off the morphia habit unless you can secure the due and complete seclusion of your patient under the care of trustworthy nurses. If she is to be treated at home (and the same remark applies to male patients), one of the conditions must be that her relations are not to see her, and that her own servants, whom she might persuade or heavily bribe to bring her morphia, are not to have access to her bedroom.

Preliminary measures.

Whatever be the line of treatment you ultimately adopt, you will find it well not to begin

that treatment at once, but to keep the patient under observation for a day or two before reducing the morphia. The organs of the body are put under severe strain during the process of suppressing the drug, and it is well that their functions should be got in order as far as may be, before that strain commences. Rest in bed, a liberal milk diet, and a calomel purge will all be beneficial, and these preliminary days will afford you a valuable opportunity of stopping alcohol, cocaine, or other drug, if the patient has fallen under such influence. This should be done at once and completely. The daily quantity of morphia is not to be diminished during these preliminary days, but the patient is to be got so far under control by regulating carefully the hours at which the injections are given. During this period also the utmost care must be taken to see that the patient has no hidden store of morphia in his clothes, about the bed, or within any kind of reach. The deceitfulness and clever cunning of the morphinist will elude any but the most extreme and minute precautions.

Suppression of morphia habit.

As to the actual suppression of morphia. This may be done in one of three ways. (1) It may be suddenly stopped; (2) it may be very rapidly

diminished; (3) it may be decreased slowly and gradually.

Sudden withdrawal.

The first method, that of sudden and complete withdrawal, brings with it great suffering and a dangerous, sometimes fatal, collapse. It should not be used unless the daily dose of the drug is very small, the habit not one of long standing, and the patient strong.

Less rapid withdrawal.

In the second method, that of rapid diminution, the procedure is somewhat as follows. If, for example, the patient has been taking 15 grains every twenty-four hours, you will give on the first day of treatment 7 grains in divided doses at those hours at which, during the days of preliminary observation, he has been accustomed to have the drug. On the three following days he will receive a grain and a half, then only a grain. Thus in some six days the morphia will have been completely stopped.

Great suffering attends this process. The patient becomes restless, then excited, shivers violently, the temperature falls below normal, there is profuse sweating and salivation, nausea, vomiting, diarrhœa, and the signs of more or less severe collapse. Sleep is impossible, and a state of more utter misery can hardly be con-

ceived. These symptoms gradually abate as the days pass, and something may be done to soften their severity while they last. The excitement and extreme restlessness may be somewhat abated by a warm bath, and by large doses—a drachm or more—of bromide of potassium. It is not advisable to use chloral, and certainly cocaine should never be employed, but sulphonal or trional, cautiously given, may help to procure sleep. During the stage of collapse you will find citrate of caffeine useful, especially when given hypodermically, and subcutaneous injection of ether may also be of service. But, if the collapse is severe, you may be compelled to give the patient an extra dose of morphia to tide him over a dangerous moment.[1]

Method by slow withdrawal of the morphia.

In the third and, I think, the best method of suppressing the morphia habit, the drug is withdrawn much more slowly and gradually, the process lasting perhaps three weeks or a month, more or less according to the quantity of morphia which has been used. The strain and suffering are considerably less than in the case of the

[1] Dr. Macleod has recently made use of very large doses of bromide of sodium in treating morphinism in China. He gives the salt in solution in doses of two drachms every two hours for the first two days, and one drachm on the third day.

second method, but, on the other hand, the discomfort is more prolonged. It is best, I think, to make each step in the reduction at intervals of about three days. This allows some sort of breathing space between each strain.

But by whatever method you proceed, you should be careful to see that your patient takes plenty of food during the treatment. Milk, raw eggs, strong soups are usually suitable, but in some cases the food may have to be peptonised. The use of alcohol should, if possible, be avoided.

Treatment during convalescence.

As the patient emerges from the treatment and his strength begins to return, he should be sent on a long sea voyage, if that be possible. In any case he will require careful watching for many months until his mental equilibrium is re-established. For at least a year his mental state is not likely to be a thoroughly sane one, and at intervals he will be seized with a craving for morphia or for alcohol. What should be specially taught him is that from time to time, particularly during the night and when the vital powers are at their lowest point, the thought of morphia and its delights will come upon him. This thought must be stamped out resolutely. At first he will experience great difficulty in

banishing the idea from his mind, but gradually the power to do this increases, and in like proportion the dangers of relapse lessen. Ultimately, as the years go on, the thought of morphia will occur to him with increasing rarity, and, when it does occur, will be banished with ease. I attach the greatest importance to this self-tuition. But unless the mind and self-control of your patient have been strengthened by healthy open-air life, this beneficial power of suppressing the thought of morphia will probably not be acquired. Without such power he will readily succumb to temptation, and will then form one of that great number of persons who, once cured, fall back into the cruel arms of the drug.

Alcohol as a poison.

Turning now to the consideration of alcohol as a poison, a few general remarks may be made before we proceed to consider the treatment to be adopted in cases of chronic alcoholism. It has already been said that one prominent and general characteristic of the action of the various toxines on the nervous tissues is that they are highly selective poisons. By this is implied that each toxine attacks with readiness certain specific sets of neurones, having probably a

special affinity for the cells of these neurones. We shall see presently which sets and linkages of neurones are specifically attacked by alcohol. But before doing so it seems necessary to emphasise another point in regard to which alcohol presents certain peculiarities not exhibited to anything like the same extent by any other poison. This peculiarity consists in the differences which characterise its action in different individuals. In the temporary intoxication of over-indulgence this diversity is very apparent. *Its selective action.*

In many persons the agreeable stimulation of alcohol is followed, when the dose is too large, by a quiet and gradually increasing blunting of the sense of external impressions and stimuli, until consciousness is lost in stupor. This may be regarded as the result of the action of a pure alcohol in a normal man. Many, however, become noisy and pass into a violent and quarrelsome excitement. This difference in mental effect may to some extent be due to impurities in the alcohol consumed, but is much more largely referable to individual peculiarity. *Its varying action on the individual.*

This condition, which we call idiosyncrasy, is in reality one arising from some inherited or *Idiosyncrasy.*

acquired weakness of certain neurones, leading to a condition of lessened resistance or of increased vulnerability. Under alcoholic intoxication one man becomes stupid, another is excited almost to mania, while in a third the sensorium may remain comparatively clear at a time when voluntary co-ordinated action of the muscles of the legs, or of those subserving articulate speech, is impossible.

And just as, in the passing intoxication of alcohol the individual weakness of special neurones mirrors itself in the clinical picture the drunkard presents, so in the less fleeting form of alcoholic neuritis similar individual peculiarities are to be observed. In most persons the motor phenomena largely overshadow the other changes, but in some the sensory predominate, in others the psychic, and in a few cases (nervo-tabes périphérique of Dejerine) the weight of the poison falls on the apparatus of co-ordination, almost to the exclusion of any other lesion.

Alcoholic neuritis. The changes found in the peripheral nerves in cases of alcoholic neuritis are, as I have taught for years, of a purely parenchymatous nature. They are not as it were local to the

nerve in question, but are expressive of a more or less severe destructive change in the whole of each neurone involved. By this I mean that the neurone suffers as a whole, cell-body, nucleus, protoplasmic processes, and axis cylinder. That the signs of degeneration are more marked towards the periphere is not remarkable, seeing that the trophic influence is less active the greater the distance from the cell. The changes which are found in the cells are, briefly, excentric movement of the nucleus and more or less complete chromatolysis. In the axis cylinders the process is one of Wallerian degeneration.[1]

Pathological changes in chronic alcoholism.

The cells chiefly affected are the motor cells in the anterior horns of the cord, the spinal ganglion cells, some cells in the pons and medulla, and the Betz cells of the cortex. In all these instances the corresponding fibres undergo more or less complete Wallerian degeneration, the whole neurone thus suffering. The mental changes which accompany and are almost characteristic of alcoholic neuritis, first described by Korsakow,[2] are no

[1] The changes are excellently described in a paper by S. J. Cole, "On changes in the central nervous system in the neuritic disorders of chronic alcoholism," *Brain* (1902), p. 326.

[2] Korsakow, "Ueber eine besondere Form psychischer Störung kombiniert mit mult. Neuritis," *Arch. f. Psych.* Bd. 21 (1890), S. 669.

doubt the clinical expression of fine changes in the cortex.

Treatment of chronic alcoholism.

The consideration of the treatment of chronic alcoholism need not detain us so long as that of the morphia habit has done. In bad cases, undoubtedly, it is best that such treatment should be conducted in those Retreats which are set apart by law for this purpose. Under the Inebriates Act of 1898 there exists the power of compulsion, and it is to be hoped that many inveterate alcoholics may in this way be rescued.

But you will encounter in the course of practice many cases in which it is not necessary or even advisable to insist on compulsory seclusion—not, at any rate, until some attempt has been made to break off the habit by less rigorous measures. In those cases in which some remnant of self-respect is preserved and some self-control exists, the cure of chronic alcoholism, in a man at any rate, may be accomplished outside of such Retreats. The personal desire to be cured must, however, be present, or no good or lasting result is possible. It is to be remembered that drinking

See also his article, "Erinnerungstäuschungen bei polyneuritischer Psychose," *Allg. Z. f. Psych.* (1891), S. 390.

convivially, even when carried to great excess, is far less dangerous than the habit of secret drinking, which is often so difficult of detection.

Suppression of alcoholic habit.

Unlike morphia, the alcoholic habit should be suppressed abruptly and at once. Although some depression follows, and even a degree of collapse may take place, these are not for a moment to be compared in severity with those which attend the suppression of morphia. They are not dangerous, and do not contra-indicate the total and immediate withdrawal of alcohol. The only exception to be made to this rule is in the case of very weak persons who show signs of cardiac failure. In these patients it may not be possible or advisable to stop the use of alcohol entirely and at once.

Treatment of shock.

The shock to the system which follows the sudden withdrawal of alcohol is to be combated by frequent feeding and careful nursing, and the sleeplessness by giving, if necessary, such sedatives as the bromides, sulphonal or trional. It is hardly needful to say that the use of morphia or cocaine is to be avoided.

Convalescence.

As the strength of your patient returns, he should be sent on a sea voyage, or should be taken to the country, in each case under proper

supervision. In the eountry he should be encouraged to take suffieient exereise to produee eonsiderable museular fatigue. This usually at onee puts an end to any sleeplessness from whieh he may have been suffering. But you should see that the exereise he takes is of a kind to interest him and to oceupy his mind. Angling, for example, is very suitable, for he who wields a heavy rod by day is seldom sleepless by night. Golf, or any other open-air pastime, involving muscular work, will do as well.

The main difficulty which you have to eneounter is not the breaking off of the aleoholic habit, but the much harder task of keeping the man straight afterwards. He is surrounded with temptations, and, among his friends who take their wine in proper moderation, he must sit a total abstainer. The moral support of a judieious friend will help him, and what I said to you just now as to the praetiee of banishing the thought of morphia applies equally to alcohol.

Syphilitic nervous affections.

Syphilitie affeetions of the nervous system are very frequent as manifestations of the aetion of that disease. The syphilitic virus is speeially prone to attack with vigour those tissues which are the

least resisting, and in a large proportion of individuals the nervous tissues present great vulnerability. It used to be held that specific lesions of the central nervous system were late manifestations of the disease, but the more recent statistics disprove this assumption. They go to show that some 25 per cent of the cases of central nervous lesion occur within the year following infection, and about 50 per cent within the first three years. I incline to think that Oppenheim[1] is correct in believing that a change has taken place in the character of the disease, and that of recent years not only is the nervous system more frequently attacked by syphilis than was formerly the case, but also that the nervous lesions occur now at an earlier stage in the malady.

Stage at which they occur.

The frequency and severity of these nervous lesions render the consideration of their treatment a specially important one, and I shall endeavour to discuss the question in some detail.

It must, in the first place, however, be frankly confessed that when once syphilis has been definitely acquired, there are no means at our disposal which will with certainty protect the nervous

[1] Oppenheim, *Die syphilitischen Erkrankungen des Gehirns*, Wien, (1903), S. 9.

tissues against its attack, nor entirely exempt the patient from those lamentable after-results which the physician so frequently encounters. We can indeed do a great deal to ward off these nervous manifestations, and without any doubt what ought to be recommended is a thorough and prolonged course of anti-syphilitic treatment. One clear deduction may be drawn from the statistics and the general experience of this unfortunate malady, namely, that it is those patients who have remained untreated, or in whom the treatment has been short and imperfect, who furnish most of the examples of the various syphilitic nervous maladies. From this it follows that the treatment should be prompt, vigorous, and thorough. As to the question of what form anti-syphilitic treatment should take, I have no hesitation in saying that, so far as lesions of the nervous system are concerned, both mercury and iodide of potassium should be employed. No doubt in some cases, such as intra-cranial gumma, the iodide gives the more rapid and brilliant results, but the effects of mercury are more stable and permanent. Both of these valuable remedies should therefore be employed, either at the same time or in sequence. Which shall be employed

Value of treatment.

Method of anti-syphilitic treatment.

first depends to some extent upon the exact nature of the case with which one is dealing. In general it may be said that it is best to commence with mercury, and to utilise the intervals between the mercurial courses to give iodide of potassium. In the case, however, of an intra-cranial gumma it is probably more convenient to commence by giving large doses of the iodide, and only after a week or two to pass on to regular mercurial treatment.

General directions.

Before considering the details of anti-syphilitic treatment, note may be made of certain general instructions and regulations which should be impressed on the patient. He should lead a very quiet life, with a reasonable amount of easy exercise in the open air. On practical as well as theoretical grounds he should refrain from the use of tobacco, and specially of alcohol. He should also avoid any considerable mental or bodily strain, very particularly that connected with sexual excess. It will be necessary to point out at some length in a subsequent chapter how great an effect is produced on the nervous tissues when two causes of disease act together. For example, a degree of pressure on the musculo-spiral nerve, which in a normal person would produce no measurable or recognisable ill effect, may in an

Avoidance of alcohol.

alcoholic give rise to severe paralysis. In this instance the action of alcohol has weakened the resisting power of the neurones and has rendered them more liable to injury. It is for this reason that it is important for the patient to avoid alcohol during the time he is under anti-syphilitic treatment. The syphilitic toxine will more easily attack neurones weakened by alcohol. It is indeed well recognised to what an extent alcoholism is associated with the disease we are considering. As a predisposing cause it is potent, partly, no doubt, because a man under the influence of alcohol is less likely to exercise self-restraint and judgment in a moment of temptation, and so more readily places himself under circumstances leading to infection than a sober man would do, but chiefly because the toxic action of alcohol gives rise to increased vulnerability of the nervous system. The prognosis of cerebral syphilis is also graver in alcoholics than it is in other men.

Avoidance of over-exertion and strain.

For a similar reason all over-exertion and strain should be avoided. The nerve cells and nerve paths which are used in such exertion become more vulnerable. Mott[1] has indeed shown

[1] Mott and Tredgold, "Some observations on primary degeneration of the Motor Tract," *Brain* (1902), p. 401.

how far even the ordinary muscular efforts of daily life are capable of determining what part of the nervous system shall be the seat of degenerative changes. A cab-driver, for example, is more apt to show changes connected with his arms, which he so constantly strains, whereas in a man who does much walking the symptoms of the disease will be more likely to appear in connection with the legs.

Details of anti-syphilitic treatment.

Coming now to note the actual details involved in the carrying out of anti-syphilitic treatment by means of mercury and iodide of potassium, we shall probably find it convenient to take a concrete example. Let us consider, then, how we should treat a specific cord affection, such as syphilitic myelitis.

Administration of mercury.

How are these remedies to be administered in such a case? Now, it is no doubt true that the action of mercury can be attained by administration by the mouth. But, in this method, if the full effect of the drug is to be obtained, one has to give doses which are very liable to produce gastro-intestinal irritation and salivation. So much is this the case that if even so mild a preparation as grey powder is given for any length of time, one has to combine some opium

with it, so as to control its action on the bowel. This addition of opium is, to my mind, inadvisable, and yet it is often necessary in order to secure a thorough mercurial action. I therefore believe that in treating syphilis of the nervous system the mercury should not be given by the mouth.[1] Two other methods are available. One may have the mercury rubbed through the skin, or it may be given hypodermically. Of these, the method by inunction is usually to be preferred.

Inunction. In such a case, then, as we have supposed, if the physician resorts to the method of inunction, he will give directions that from 60 to 120 grains of mercurial ointment be rubbed into the skin daily, a different part of the body being selected on each occasion. The patient may begin, for example, with the right arm, then the left arm, the right thigh, the left thigh, and then *da capo*. A square of linen rag is taken, sufficiently large to cover amply the area of skin to be rubbed, and on it a portion of the quantity of ointment

[1] Save in very exceptional cases. No doubt this is to some extent a counsel of perfection in the sense that, while certainly inunction is the better method, cases come before one in which, for private reasons, the patient does not see his way to perform the manipulations required for proper inunction. In cases of this kind the mercury may have to be given by the mouth.

to be used is placed. The patient is instructed to rub the skin vigorously with this, adding from time to time a little more of the ointment until the whole quantity has been rubbed in. This operation should take from ten to fifteen minutes. Thereafter the square of rag is to be laid over the skin surface, covered with a layer of cotton wool and retained by means of a light bandage. The inunction is to be made at night, and the patient sleeps with the rag and bandage so adjusted. In the morning the vigorous application of soap and hot water will remove any traces of unabsorbed ointment. It will be found convenient, when ordering the mercurial ointment, to instruct the chemist to send it in a number of little packets, enclosed in waxed paper, each containing the quantity necessary for one inunction.

In certain Continental watering-places, where the treatment of syphilis is specially attended to, the inunctions are made by means of a glass instrument, shaped like a handle and bearing on one end a hemispherical surface of ground glass, with which the ointment is rubbed in. The inunctions are made by a skilled rubber, about an hour after the patient has left a warm

bath. I think, however, that the method of inunction which I described first is the better for such cases as those we are now considering.

Course of inunction.

Such inunction as this should be made daily for a fortnight, or, if possible, three weeks. There should then come a pause of a week, and, if no stomatitis has developed, a new series of frictions may then be entered on. After the second series of inunctions is completed, a pause of a month's duration is advisable, after which the treatment should be recommenced. To be complete, these series of inunctions should extend (with intervals of increasing length) over a period of at least three years. There are indeed some who recommend that during each subsequent year, up to eight years from the date of infection, the patient should annually undergo a cure of seven weeks' duration. The necessity of taking care of the teeth and gums during such treatment as this is well recognised. The teeth should be carefully brushed after every meal, and the mouth and throat washed out with chlorate of potash solution.

Hypodermic administration.

While I believe that as a rule the method by inunction is to be preferred, there are some cases in which it may be advisable to administer the

mercury hypodermically. When the symptoms are severe, and the need for mercurial action is pressing, then the subcutaneous method, which is more rapid and energetic in action than inunction, is to be preferred. It is not, however, without its inconveniences. It is often painful, and, if due care is not taken, much induration and even abscess formation may result.

The syringe to be used must be carefully sterilised. The needle, which should be of platinum, is to be heated in a flame just before use, and cooled in carbolic oil. The injection is best made in the gluteal region, and the solution should be injected deeply into muscular fibre. The skin is to be carefully cleansed before the operation.

Preparations for that purpose.

Various preparations of mercury have been employed for hypodermic administration. Grey oil has been much used, as well as calomel in vaseline, but neither of these preparations is to be recommended.

The red iodide (mercuric iodide) is frequently employed for injection. With the aid of a little iodide of sodium it is readily dissolved in the proportion of one grain in 64 minims of distilled water. Of this solution from two to six minims

Panas' solution. may be injected. An even better preparation is that of Panas :—

Mercuric iodide, 0·4 grm.
Sterilised olive oil, 100 c.c.

The oil is purified by washing with distilled water, and, after separating it, is again washed with absolute alcohol. It is finally sterilised. Of this solution 1 c.c. (nearly 17 minims) contains about 4 milligrammes ($\frac{1}{16}$ grain) of mercuric iodide.

Delpech's solution. Delpech's solution for hypodermic use, which is much employed in Paris, consists of an ammoniated peptone of mercury of the following composition :—

℞ Hydrarg. perchlor.	10 grammes
Peptone (dry)	15 ,,
Ammon. chlorid.	15 ,,
	Misce.

This preparation is to be dissolved in distilled water, or preferably in glycerine, in the proportion of 40 centigrammes in 30 grammes.

There is, finally, sozo-iodol-mercury, a preparation which has recently come into some vogue for intra-muscular injections in syphilis. Of this substance $2\frac{1}{2}$ grains are to be dissolved, along with 5 grains of sodium iodide, in 100

minims of distilled water. The resulting solution may be given hypodermically in doses of 10 minims.

During the greater part of this period iodide of potassium should also be given, the dose depending on the individual, but being in any case as large as can be borne. It is well to begin with 30 or 40 grains in the day, and rapidly to increase the dose as far as seems advisable in the particular case. To get the full effect of the iodide it is sometimes necessary to give very large doses —150 or 200 grains in each twenty-four hours. *Iodide of potassium.*

In cases in which the iodide has to be administered for long periods, use may be made of a preparation called iodalbacid by its discoverer, Dr. Blum of Frankfort. It is an iodine-albumin (analogous to iodo-thyrin), formed by the action of iodine on moist albumin. When taken internally it readily breaks up, liberating its iodine (of which it contains about 10 per cent), and not producing the disagreeable effects sometimes seen after the exhibition of potassium iodide. Iodalbacid should not be used in cases in which a rapid action is desired. In these, iodide of potassium is to be prefered. But for the more chronic cases it has been found very useful. It *Iodalbacid.*

is freely soluble in water, but is best given either in cachets or in the form of tablets. The quantity which should be administered daily is from three to six grammes (45 to 90 grains), in divided doses. I have seen excellent results from the use of this preparation. It is not nearly so liable to give rise to symptoms of iodism as are the ordinary salts of iodine.

Iodopin

Of iodopin I have had little experience, but in the hands of many observers it appears to have proved a valuable preparation.

Surgical measures.

In the case of syphilitic gummata lying on the motor cortex which have resisted anti-syphilitic treatment, it is sometimes necessary to call in the aid of the surgeon in order to their removal. To this question attention will be directed in a future lecture.

Para-syphilitic maladies.

As to the para-syphilitic maladies, and in particular locomotor ataxia, the experience of most physicians is that the results of anti-syphilitic treatment are usually unsatisfactory.

Tabes.

What I have myself seen is, for the most part, in corroboration of this view. At the same time one cannot altogether overlook the results of Erb, who strongly recommends this procedure. On the whole, except in cases in which there is

commencing optic atrophy, and in those in which I have reason to be certain that such treatment has already been efficiently carried out, I incline to give a thorough course of mercury. There can of course be no hesitation in advising such treatment in cases that show definite signs of syphilitic lesion, nor in those in which the infection is of recent date. There is always, in cases of tabes, need of more than usual care in watching the patient while he is being placed under the influence of mercury. The weight of the patient should be recorded from time to time, as well as the condition of his blood, and a sedulous watch should be kept on the state of his digestive organs. In this way the physician will receive due warning in those cases of tabes in which prolonged mercurial treatment might be productive of harm.

Anti-syphilitic treatment in that disease.

Precautions.

The objection which many have to the employment of mercury in cases of tabes does not apply to iodide of potassium. That drug may be freely given, and appears sometimes to hold in check the degenerative changes on which the symptoms of the malady depend.

LECTURE III

TREATMENT OF PERIPHERAL PARALYSIS

Definition of term "peripheral paralysis"—Anatomical arrangement of the lower motor neurone—Diseases which affect it—Their symptoms—Treatment of peripheral paralysis—Removal of cause—Local measures—Use of drugs—Use of galvanic current—Use of faradic current—Massage—Exercise—Tendon-transplantation.

Definition of term "peripheral paralysis."

It is needful, in the first place, to define with precision what is meant by peripheral paralysis, seeing that that term is, unfortunately, used by certain authors with some looseness and inaccuracy. It is sometimes taken to mean a paralysis resulting from a lesion peripheral to the spinal cord, *i.e.* of motor nerves. This is obviously an unscientific definition. The term peripheral paralysis, as I shall use it in these lectures, includes all forms of paralysis resulting from lesion of the peripheral motor neurone, whether that lesion lie in the centres in the cord, or peripheral to these centres. The same observation applies, of course, to the motor cranial

nerves. The physiological functions of this lower motor neurone impress a special character on the symptoms which result from its destruction, and these symptoms are in their essence the same, at whatsoever point in the neurone the lesion may be situated. They differ of course in detail, and do so sufficiently in many cases to allow a localising diagnosis to be made. But, broadly speaking, the phenomena are essentially the same, and hence I hold strongly that the term peripheral paralysis should always be used according to the definition I have given you.

Arrangement of lower motor neurone.

As you are aware, the lower motor neurone consists of the nerve-cell lying in the anterior cornu of the spinal cord, or in the corresponding medullary or cranial nuclei, with its protoplasm, its nucleus, and the arborisation of its dendrites, of its axis-cylinder process or axone, prolonged to become the efferent nerve-fibre, and of its terminal arborisation connected with the substance of the muscular fibre which it innervates.

These axones, passing from the cord, go to form the anterior root. They proceed for the most part from cells lying on the same side of the cord, though a few appear to pass by the

anterior commissure, and to be the axones of cells lying in the anterior horn of the opposite side. The large motor cells of the anterior horn are arranged in certain groups, a median group, a lateral posterior, a lateral anterior, a median posterior, and a median anterior group. We do not know exactly which muscles each of these groups represents, but as the result of clinical observation, and by noting the position of the cell atrophy which has been found after various amputations and the like, it has been possible to gain some rough idea of the physiological arrangement.[1] When we come to consider the treatment of acute anterior poliomyelitis we shall see the importance of this arrangement of cell groups.

Grouping of cells in the cord.

Diseased processes may strike at the whole or at any part of this neurone, and may be of very varying character. Any lesion involving the anterior horns of the cord (or the corresponding motor cranial nuclei) which destroys the cell, destroys at the same time the whole neurone, because the cell is its trophic centre. It produces for a like reason rapid atrophy in the

Diseases which attack the neurone.

[1] See paper by Lapinsky, "Ueber die Lokalisationen motorischer Funktionen im Rückenmark," *D. Arch. f. Nervenheil.* Bd. 26 (1904) S. 457.

muscle. Or it may be that the axone running in the peripheral nerve may alone be affected, the result usually of pressure, wounding, or other local cause. Or, finally, the neurone may be attacked as a whole by the toxines of disease, by the products of faulty metabolism, or by some metallic, alcoholic, or other extraneous poison.

But whether it be the neurone as a whole which is attacked, or the cell in the cord, or only the motor nerve in its course, the result as to motor symptoms is, in essentials, the same. And these motor phenomena, familiar to you from the study of many such cases, are briefly as follows :—

Signs of peripheral paralysis.

The muscles innervated by the affected neurone are removed from the influence of the will : they are paralysed. And, owing to the fact that their trophic centres lie in the cells of the lower neurone, the muscles rapidly atrophy, and for a like reason they lose their tonus and become flaccid. The reflex loop being destroyed by the lesion in the neurone the deep reflexes are lost. And, finally, there is present that curious phenomenon first described by Erb, the reaction of degeneration. This reaction, highly im-

portant in regard to diagnosis and a valuable guide to prognosis, is also useful, as you will presently see, in relation to treatment. Stated briefly, its features when fully developed are as

Reaction of degeneration.

follows. The nerve when stimulated by the galvanic or by the faradic current gives no response, *i.e.* no muscular contraction follows. Nor does any contraction result from the application of a faradic stimulus over the muscle itself. But when the muscle is stimulated by the galvanic current, it contracts,—often indeed it responds in this way to a current too weak to give rise to contraction under normal conditions. And the muscular contractions so produced are sluggish, and not sharp and almost instantaneous as in health. Also you will frequently find that the anodal closing contraction is more readily elicited than the cathodal, a condition opposite to that which obtains in health. In

Polar changes.

fact the polar conditions are often so altered that we get, in order of current strength :—

(1) A.C.C.
(2) K.C.C.
(3) K.O.C.
(4) A.O.C.

instead of the normal—

(1) K.C.C.
(2) A.C.C.
(3) A.O.C.
(4) K.O.C.

But while these may be taken as the characteristics of the reaction of degeneration, and the most important of these is the sluggishness of the response to galvanism, a lighter and less fully developed form of the reaction is often met with, in which the faradic irritability, though diminished, is not completely lost, and in which the nerve still conveys a galvanic stimulus. You will presently see the importance, from the point of view of treatment, of recognising this lighter or partial form of the reaction of degeneration.

Partial reaction of degeneration.

Turning now to the consideration of the treatment of these forms of disease, I have first to tell you that, in many cases, paralysis of the lower neurone is very amenable to treatment. You will sometimes get results which are surprisingly good, more so, I think, than in almost any other form of nervous disease.

Treatment of peripheral paralysis.

In the first place, then, having made your

diagnosis, and having satisfied yourself that the lesion with which you are dealing is one of the lower motor neurone, it is your next duty to *Causation.* ascertain, as nearly as may be, the cause which has acted in the individual case you are studying. In many cases, indeed in the large majority, you will be able to do this with a fair approach to accuracy. If the neurone is affected as a whole, you will look for evidence of the presence of toxines of infective disease, of altered metabolism and auto-intoxication as in diabetes, of metallic poisons such as lead, arsenic, mercury, or of alcohol. If, on the contrary, the lesion is one acting on the trunk of the nerve, you will search for such traumatic causes of neuritis as pressure, tearing, wounding, and the like.

And, if you are so successful as to discover it, the first step in treatment must necessarily be *Removal of cause.* an attempt to remove that cause. Now I have, in the last lecture, spoken in some detail of the means by which the removal of the toxines of disease and other nerve poisons should be attempted, or by which their deleterious action may be combated. To this we need not now return. Something may, however, be said as to the treatment of acute anterior poliomyelitis

Acute anterior poliomyelitis.

during the febrile stage. We possess, unfortunately, no antitoxine for this complaint, though it may be that the future will bring us one, and therefore we cannot directly counteract the toxine which is at work. We can, however, do something towards eliminating the poison, and for this purpose we should see that the bowels are kept well open, preferably by means of calomel, and that a somewhat profuse diaphoresis is at once established. The method of lavage of the tissues by means of rectal irrigation formerly described (see p. 49) might usefully be employed. But indeed it is almost superfluous to speak of such methods, for only very rarely can a diagnosis be made during this early stage of the malady. When we are able to reach a positive diagnosis, the mischief has been done, and the lesion in the grey matter is complete. That lesion is, however, as you know, one which tends to limit itself, and while cells in the centre of the lesion are permanently destroyed, those lying towards its periphere tend to recover. We can, to a certain extent, promote that recovery by giving iodide of potassium. This hastens the absorption of the inflammatory exudation. Now I have already alluded to the salts iodine,

and I shall have to refer very frequently in the course of these lectures to the beneficial action they produce in nervous disease. Something may, therefore, be said now as to the method of that action. It may be confessed at once that we do not know exactly how these iodine salts act. Probably, indeed, their action is manifold. But in many cases I believe that the good effects which follow the administration of these salts are the result of the stimulus to phagocytosis to which they give rise. The experiments of Heinz[1] point strongly, though not quite conclusively, to that conclusion; but these results, taken along with the outcome of clinical experience, warrant us in believing that an increased phagocytosis is the important factor in the action of iodide of potassium. This is, no doubt, aided by the effects that salt produces on the blood-vessels. We shall have to consider this point more fully in speaking of cerebral hæmorrhage.

Mode of action of iodide of potassium.

Traumatic cases.

In traumatic cases, where the nerve has suffered from compression or tearing, the result of fracture of bone, of dislocation, tumour,

[1] Heinz, "Ueber Jod und Jodverbindungen," *Virch. Arch.* Bd. 155 (1899), S. 44.

inflammation of neighbouring parts, or the like, surgical aid is called for. But even if it be possible to remove the cause of the paralysis by surgical measures, the injured nerve is not necessarily thereby restored to health, and much careful treatment will usually be found needful before its function is regained. It is hardly necessary to say that in the case of a wound severing a nerve, the cut ends should be united as soon after the injury as possible.

The importance of rest in treatment of these *Rest.* paralyses of the lower neurone is very obvious. The patient should be confined to bed until all acute symptoms have completely disappeared. For any movement involves some attempt, at least, to set up functional activity in the neurone, and this is not desirable so long as an acute process is going on. In acute anterior poliomyelitis, for example, it would be very harmful in the early stage to stimulate those nerve-cells which are within the diseased area, and in that affection, as well as in multiple neuritis, a single exertion may do great damage to the cells and fibres implicated. But not only is it very harmful to attempt to set in activity these diseased structures during the acute stage of the affection,

but in multiple neuritis, as well as in traumatic neuritis, the contraction of the muscles through or between which the affected nerves pass, may produce such compression as may be very detrimental to recovery. For these and for other similar reasons confinement to bed should always be enjoined, and the patient be instructed to keep the affected parts as still as possible.

Blood-letting. The abstraction of blood by means of leeches or wet cupping is sometimes of considerable service at the outset in cases of rheumatic facial paralysis and other local inflammatory lesions of *Heat.* peripheral nerves. In the same class of cases hot poultices and fomentations, applied over the inflamed region of the nerve, have a soothing effect, and probably mitigate the severity of the process. You will also find that hot poultices applied to the spine over the seat of the lesion are very grateful to the child suffering from acute poliomyelitis. In cases of local inflammatory neuritis, when the acute stage has passed away, it is sometimes advisable to apply fly-blisters, or even the stronger counter-irritation of the cautery over the seat of the lesion. But if you are going to *Blisters.* apply a blister in such a case, you should be careful not to do so at a point where there is

anæsthesia, for in all probability the nutrition of the skin will be so lowered in that area that you may have great difficulty in getting the blister to heal. In multiple neuritis these external applications are not nearly so useful as in those conditions of which we have been speaking.

Medicinal treatment.

The treatment by drugs is not very successful in cases of paralysis of the lower neurone. In all instances tonic treatment with iron, cod-liver oil, strychnine, quinine,[1] etc., will be required in the later stages. But it is difficult to obtain any very decided medicinal action on the progress of the disease itself. Iodide of potassium, sometimes useful in the chronic stages of multiple neuritis, is undoubtedly of service in localised inflammation of a nerve trunk, and its utility is increased, even in non-syphilitic cases, by combining with it small doses of mercury. I have already alluded (p. 102) to the probable mode of action of iodide of potassium in such cases. You will find salicylate of soda of use in cases of rheumatic neuritis, and it should be tried in every case of lesion of the lower neurone where the

[1] The use of arsenic as a tonic in these cases is very questionable, because it is itself capable of giving rise to neuritis, even in comparatively small doses. If it is employed, its action should be watched with great care. For the same reason the doses of mercury given should be small.

attack seems to have been brought on by exposure, and especially when there is any suspicion of a rheumatic tendency in the patient. Antipyrine and its allies may also be of service under these conditions. I have seen several cases in which small doses of phosphorus appeared to do considerable good.

Strychnine.

Its use in progressive muscular atrophy.

Strychnine is undoubtedly of great use in some cases of progressive muscular atrophy. Gowers, who relies upon it in this affection, holds that it arrests the disease in more than one half of the cases. I fear this is rather too optimistic a view; at any rate my own experience does not bear it out. But I have seen excellent results in some cases, and it should certainly be tried. In so hopeless a disease as progressive muscular atrophy one is indeed glad to grasp at any straw and to employ any method which seems to promise even slight benefit. The strychnine should be given hypodermically, and the quantity, at first small ($\frac{1}{50}$ grain of the nitrate of strychnine), should be rapidly increased to the limit of tolerance. This dose should be administered once a day, and the treatment should be continued for many months. Strychnine sometimes appears to do good in acute polio-

myelitis. In such cases it should be given by the mouth and in very small doses, and great care should be taken not to begin its administration until six weeks or two months have elapsed since the commencement of the disease.

Bulbar paralysis. In cases of bulbar paralysis, also, you may find that strychnine is of benefit. The prognosis in that disease is, as you know, very grave, and we can hardly hope to be able to do more than to prolong life a little and render the condition of the patient more bearable. *Its general management.* In it, as in progressive muscular atrophy and in amyotrophic lateral sclerosis, it is very important that the neurones which have become involved should be used as little as possible. The patient suffering from bulbar paralysis should therefore not be encouraged to speak more than is necessary. His diet should be such as to involve no chewing, *Diet.* and should be selected with a view to ease in swallowing. It should, therefore, consist of semi-solids and of liquids, such as raw eggs, milk, strong soups and the like. As the disease progresses recourse must be had to the stomach tube in administering food. The distressing salivation is best controlled by atropine along with morphia.

Myasthenia gravis.

Very much the same treatment is advisable in cases of myasthenia gravis. In that disease, however, the necessity of rest, both bodily and mental, is greater. An equal degree of care should be used in regard to feeding. The use of strychnine in myasthenia gravis is not advisable.

Multiple neuritis.

In multiple neuritis also, once the acute stage is over, strychnine is a very useful remedy to aid in restoring the tonus of the wasted muscles. It should be given hypodermically in doses, at first small, then gradually increasing, and its use should be continued for many weeks at a time.

The cardiac weakness which accompanies some forms of multiple neuritis, particularly that due to diphtheria, may require the use of special remedies—alcohol, digitalis, strychnine, camphor, musk, etc.

Electricity.

Electricity is one of the most important therapeutic means at our command in cases of paralysis of the peripheral neurone, and you have had many opportunities of observing and of estimating the benefits which follow its employment.

For the moment we shall exclude from con-

sideration the action of electricity on the sensory nerves, to which our attention will be subsequently directed, and discuss only its therapeutic action on the lower motor neurone and upon the muscles which that neurone innervates.

The whole subject is exceedingly difficult and obscure, resting as it does in part on exact experiment, in part on empiricism ; but I think you may take it that there are, speaking generally, two main ways in which electricity may be usefully employed in such cases as those we are now considering. In the first place, where a motor nerve no longer transmits voluntary impulses, you may endeavour to improve its nutrition and restore its function, and for this purpose you will use the galvanic current in the manner to be presently described. In the second place, you may have to stimulate to contraction those muscles which have been cut off from their trophic centres in the cord, and which are consequently wasting. For this purpose you may sometimes employ the galvanic current, but you will find that the faradic current is more generally of service.

Experimental results.

Dealing now with the first of these methods, I may remind you of the experiments of E.

Remak, which go to show that the passage of a galvanic current through a motor nerve produces an increase of excitability in the nerve so treated —an excitability which persists for some time after the current has ceased to pass. You also remember the often-quoted experiment of Heidenhain, by which he showed that when a muscle-nerve preparation has been so damaged that it will no longer respond to the opening or to the closing of a comparatively strong current, you are able, provided the damage has not been too great, to restore its function by passing through it a galvanic current, in an ascending direction, the current being continued for a longer or shorter time. Of the accuracy of these and many similar results there cannot be the slightest question. When you come, however, to consider the clinical aspects of the matter you will not find things so clear.

Electrotonic action.

The good results of the application of a galvanic current to a motor nerve which is no longer conducting voluntary impulses, consist partly in its electrotonic action; that is, in those obscure changes which show themselves in an increase of excitability at the negative pole and

a decrease at the positive. They are also due to that combination of effects called by the elder Remak the catalytic action of the current. This term—not to my mind a good one, but commonly employed—may be taken to include the electrolytic action, that molecular dissociation and rearrangement which takes place in the tissues at and between the poles, as well as the cataphoric action of the current by which an osmotic flow is set up in the fluids of the tissues in the direction from the positive to the negative pole. To this may be added the action of the current on the blood-vessels supplying the nerve, whether that action be on the fibres of the vascular wall or on the vasomotor nerves. *Catalytic action.*

In these and probably in other ways the nutrition and activity of function of a motor nerve may be deeply affected by a galvanic current, and there cannot be any doubt that most favourable results are produced by this form of electrical treatment in many cases of peripheral paralysis.

These results on the nerve, which we have been considering, are obtained, you will observe, during the passage of the galvanic current, and

are to be carefully distinguished from the stimulating and exciting action which takes place when the current is made or is broken. You should indeed, for the purpose we now have in view, avoid as far as possible these stimulating effects, and therefore it is best (if you can make the arrangement) to put on the current by means of a rheostat, after the electrodes have been applied, so that it may be slowly and steadily increased to the amount you require, and at the end of the sitting as slowly and steadily cut off. In this manner any rapid oscillations are avoided. The current employed should not as a rule exceed 6 milliampères (indeed 2 to 3 m.a. may often be sufficient), working with electrodes measuring from one to two inches in diameter. The length of time during which the current is allowed to pass should be about five minutes, more or less, according to the acuteness of the case, and the application may be made daily or every second day. You will find it best to place the cathode or negative pole as nearly as may be over the injured part of the nerve, while the anode is applied to an indifferent point, such as the sternum. It is hardly necessary to remind you that the electrodes should be

Method of using galvanic current.

thoroughly soaked in warm saline solution before being applied to the skin. You should watch the galvanometer very carefully during the application of the current, for the resistance of the skin rapidly diminishes as the current passes, and it may, in consequence, become necessary to diminish the amperage.

Effects.

Using the galvanic current in this stable way, you may expect to see some immediate improvement in favourable cases. During its application the patient will probably experience some slight return of voluntary motor power, the conducting power of the nerve having been more or less restored. If the lesion is recent and the case a favourable one, you may obtain complete restoration of motility after three or four applications of the current. I have seen cases of well-marked facial paralysis recover in this rapid way. In other cases it may require twenty sittings or more before so favourable a result is reached. In any case the cure will be accelerated in its later stages by making use of the stimulating power of the galvanic or of the faradic current in the manner to be presently described. It is needful, however, that you should fully understand that these good results—and they

Favourable cases for its application. are sometimes brilliant in their success—are only attainable in the lighter cases, when the lesion lies in the course of the nerve, and when the reaction of degeneration is only a partial one. If the fibres of the neurone are actively degenerated, you will not obtain any such distinct success. But even in these it is well to apply the galvanic current occasionally, say once every four or five days, for it is exceedingly likely (though there is no absolute proof of this) that the current exerts a favourable influence on the process of regeneration of nerve fibre. The earlier a case comes under treatment the more successful that treatment is likely to be, but even in cases of some standing the results may be good.

From what has been said it will be clear to you that this form of treatment is useful in cases of paralysis produced by localised lesion of peripheral nerves. In the so-called rheumatic *Facial paralysis.* facial paralysis most successful results may be obtained, especially if the case be seen early. The cathode (about one inch in diameter) should be applied to the mastoid or over the point of exit of the nerve, the anode being placed on the sternum, and a current of from 1 to 3 milli-

ampères applied. The sitting should not, as a rule, exceed five minutes in duration.

Musculo-spiral paralysis.

Lesions of the nerves of the limbs are also, as a rule, very amenable to this form of treatment; indeed it was in connection with musculo-spiral paralysis that E. Remak[1] published those statistical results which have enabled us to appreciate so distinctly the value of galvanic treatment in cases of paralysis of the lower motor neurone. In such cases the electrodes should be two to three inches in diameter. The cathode should be placed over the seat of the lesion, the anode over the sternum, and a current of from 4 to 6 m.a. should be employed.

Multiple neuritis

From what has been already said you will gather that when the paralysis depends on parenchymatous degeneration of the nerve fibre, in toxine poisoning, in multiple neuritis, no such successful results are to be looked for. Indeed in cases of this form of neuritis it is not advisable to employ electricity during the early part of the acute stage. But when that stage has begun to subside the galvanic current may be usefully applied, provided that a rheostat be used and

[1] E. Remak, "Ueber die antiparalytische Wirkung der Elektrotherapie bei Drucklähmungen des N. radialis," *Deutsche Zeit. f. Nervenheilkunde*, Bd. 4 (1893), S. 377.

that the opening and closing shocks be carefully avoided. Currents of 2 to 3 m.a. with electrodes of about two inches in diameter are sufficient, and the anode may preferably be applied over any tender nerve trunks. Considerable good may in this way be effected and the process of regeneration hastened.

Acute anterior poliomyelitis.

Still less may we expect success from this form of treatment in those cases in which the lesion lies more particularly in the anterior horns of the cord. It is sometimes used in infantile paralysis, but I do not think that in that disease or in the more chronic forms of poliomyelitis or in progressive muscular atrophy you will produce any distinctly beneficial result. Perhaps an exception to this may be found in the case of bulbar paralysis, in which disease the stable application of the galvanic current occasionally appears to do some good. The electrodes, of considerable size, should be applied over the two mastoid processes, and a current of 1 to 2 m.a. employed. It is particularly important to make use of a rheostat in these cases, and to avoid all rapid variations of current.

Turning now to the other form of electrical

treatment in peripheral paralysis—that, namely, which depends on stimulation of nerve and muscle—I have first of all to tell you that, while this is of great service in many cases, it may be safely used only when the acute stage has completely passed away. A certain amount of such stimulation may be brought about by the so-called labile use of the galvanic current—that, namely, in which, while one electrode is kept stationary, the other is slowly moved over the nerves and muscles. For this purpose a roller electrode may be employed. The movement of this from point to point, together with the fact that the area of the electrode in contact with the skin varies somewhat according to the compressibility of the tissue on which it happens to rest at the moment, renders the current unsteady in amount, and this produces a stimulating action on nerve and muscle.

Stimulating effects of currents.

Labile application.

A more distinctly stimulating effect is, however, produced by making and breaking the galvanic current, or by means of faradism, or by combining both forms of current; and muscular contraction may be brought about either by placing both electrodes directly over the muscle or by stimulating the nerve at its motor point.

The muscular contraction so occasioned, when produced in muscles which are largely or entirely incapable of voluntary movement, is very useful in keeping up their nutrition and tonus. The blood-flow in the contracting muscle is accelerated, and the mere mechanical pressure exerted on neighbouring veins and lymph channels tends to prevent stagnation in these vessels.

Faradic current. But, in addition to this, the faradic current appreciably affects the conductivity of the motor nerve through which it passes. If you stimulate such a nerve with a current a little weaker than is required to cause muscular contraction, you will find that after the current has been passing for a little time the excitability of the nerve has been so increased that muscular contraction commences. And it is found by experiment that this excitability persists for some time afterwards. But you must not suppose that by the strongest faradic stimulation of a motor nerve above the seat of lesion you can break through the barrier which is opposing motor conduction. That is not possible, and to attempt to do it forcibly may be very harmful.

Reflex effects. And there is yet another point which you must not forget, namely, that by the stimulation

of the sensory nerves which the faradic current (particularly the faradic brush) produces, the motor fibres may be reflexly affected. For, in the case of diseased processes in mixed peripheral nerves you have to remember that as a general rule the motor fibres suffer most, and that it is usual to find that, though no efferent impulses can pass down the nerve, afferent sensory impressions are still conveyed upwards. The powerful stimulus of the faradic brush, transmitted in this way to the centres, gives rise to centrifugal impulses down the motor fibres, and although these cannot break through the obstacle to motor conduction if the paralysis is complete, yet they are of great benefit, when regeneration is in progress, in completing the cure in cases of peripheral paralysis.

Exhaustion produced by faradic current.

In stimulating the muscles to contract, in these peripheral cases, by means of the faradic current, you should bear in mind that you may readily produce considerable exhaustion. Physiological experiment has shown that the exhaustion of motor nerves when faradised increases with the frequency of the interruptions, and you will therefore arrange your apparatus so that the making and breaking in the primary coil are not

too rapidly effected. Once a second is about the rate at which I like to see the hammer vibrate, and you may find that an even slower rhythm is sometimes advisable. As to the strength of the current to be employed, you may take it that what will give a distinct and good muscular contraction is sufficient. More than this is not necessary and, indeed, very strong stimulation may be distinctly harmful.

Neuritis. In neuritis resulting from inflammation of or pressure on the nerve trunk, when the chronic stage is reached, much good may be done by means of electrical stimulation. The galvanic current with slow interruptions may be used, the strength being just sufficient to cause muscular contraction, and it should be remembered that if the reaction of degeneration be present, it may be necessary to use the anode in order to obtain such contraction. If the muscles can be made to contract with faradism, that form of current is still more useful. I have seen much benefit result from faradic treatment even in inveterate cases. But you should lay it down to yourselves as an absolute rule that such stimulation is not to be used if any pain remain after its application, and, further, that on the appearance of any

contracture all stimulating electrical treatment should at once be stopped.

Multiple neuritis.

In multiple neuritis, when the acute stage has completely passed away, galvanic stimulation of nerves and muscles may usefully be employed, and, somewhat later in the case, the faradic current may be made use of. But here also care should be taken that no after-pain results.

Acute anterior poliomyelitis.

In acute anterior poliomyelitis no electrical treatment will do good in the case of those cells and fibres which have undergone complete degeneration, nor in that of the muscles the trophic centres of which are thus destroyed. But there are always cells and nerve fibres in which, though their function is affected, the process has not gone its full length. In these and in the corresponding muscles partial if not complete recovery is still possible under suitable treatment. For such nerves and muscles electrical stimulation is of considerable value, but it should not be commenced before the second or third week. It is best to employ galvanism in the first instance; indeed you may find in many cases that it is only by galvanism that the muscles can be made to contract. Later in the case faradism is more

useful. But, inasmuch as these cases occur in children, you have to be very careful in so using electricity as not to frighten your patient, and it is necessary to commence with very weak currents, or indeed to use no current at all, until the child has become accustomed to the application of the electrodes. The strength of current may then be gradually increased, but it should be at no time greater in amount than that just necessary to cause distinct muscular contraction. In these cases, however, massage and, still more, carefully devised exercises, produce a better effect in restoring muscles which have been partially paralysed than does electricity. We shall consider presently how these aids to recovery are best applied.

Progressive muscular atrophy.

In progressive muscular atrophy, on the other hand, very little good is effected by the use of electricity; but, nevertheless, it is right to give that form of treatment a trial in every case, using faradism if the muscles can thereby be made to contract, or if not, galvanism. But you should see that the current employed in either case is a weak one, for undoubtedly strong stimulation does harm in this disease. In bulbar paralysis some little benefit may follow faradisa-

tion of the lips, tongue, and palate, but here also the currents used should be very weak.

Massage.

Too much prominence can hardly be given to the benefits which result from massage in cases of paralysis of the lower motor neurone. By this means of treatment, when rightly conducted, the nutrition of the tissues, and particularly of the muscles, is greatly improved, and the return of venous blood and of lymph accelerated. How much such stimulus to blood-flow is required, is shown by the bluish tinge and the low temperature of the paralysed limb.

Method of employment.

In all forms of neuritis and acute poliomyelitis massage is very useful. In no case, however, should it be employed until the acute stage is well over, and in neuritis care should be taken that the rubbing is not made over inflamed nerves. The treatment should be begun gently, light friction being applied in the first instance. Later on in the case kneading movements may be made use of, which, in cases of traumatic and pressure neuritis may ultimately be performed with the full force of the operator. I have repeatedly seen great good result in this affection from massage which was distinctly painful. In multiple neuritis

and in poliomyelitis any rubbing or kneading which causes pain should be avoided.

A light and gentle massage may be tried also in cases of progressive muscular atrophy. Some little benefit may be thus obtained in that most hopeless malady.

In cases of poliomyelitis the affected limb should be stroked with the flat of the hand—always in an upward direction—over its whole surface. Then, grasping the affected muscles with the whole hand or with both hands, but not with the fingers only, the operator should knead and roll these muscles thoroughly. Going over the whole of the affected muscles methodically, a quarter of an hour or half an hour may thus be spent, but fatigue on the part of the patient is to be avoided. The process should be repeated at least once a day. In this way the nutrition of the muscles which have suffered may be greatly increased, and in some cases they may even be brought back to a normal condition. The limb should be kept warm by being carefully enveloped in flannel. Any pressure which might interfere with circulation ought, of course, to be avoided.

In all cases in which certain muscle groups in a limb are paralysed while others escape, the

sound muscles, being no longer opposed by their paralysed antagonists, tend to shorten and to pass into a state of contracture. This shortening is, of course, accompanied by a corresponding stretching and lengthening of the paralysed muscles. If you think for a moment of a case of "wrist-drop," from whatever cause, you will remember how shortened and contracted the flexors had become and how over-stretched the extensors. Now it has been found that a muscle which would otherwise recover will not readily do so, so long as it is over-stretched and lengthened. You must relax its fibres as much as possible before your treatment will have its proper effect. Therefore, before commencing to apply faradism or massage to the paretic extensors you should first overcome the contracture of the flexors, cause these to lengthen, and in this way relax the extensors and allow them to contract. This is best done, in the case of "wrist-drop," by applying a light iron splint to the flexor aspect of the forearm and hand. This splint should be gradually bent more and more, opposite the wrist, so that the hand is brought first into the line of the forearm and, ultimately, as the contracture of the flexors is overcome, into

Conditions of contracture and of over-stretching.

Contracture must be overcome.

Method in cases of "wrist-drop."

a position of hyper-extension. The extensors are thus relaxed and allowed to shorten. In this condition they may be stimulated by faradism and carefully kneaded and beaten. Recovery of muscles which have been paretic for years may thus be brought about. The same principle applies to all cases of paralysis from lesion of the lower motor neurone where, from unopposed action, a sound muscle group has passed into a state of contracture.[1]

There remains for consideration the most powerful stimulus of all and the most natural, *Exercise.* namely, Exercise. When the muscles which have been paralysed begin to recover the power of voluntary movement, when the motor fibres begin again to conduct impulses from the cortex, such voluntary movement should be encouraged. As the days pass you may add to the usefulness of such voluntary movements in promoting the return of power, by opposing resistance with your hand to such movements—a resistance which should be gradually increased from day to day. *Gymnastics.* A little later in the case gymnastic exercises may

[1] This method is well described by Tubby and Jones, *Modern Methods in the Surgery of Paralysis*, p. 46. London (1903).

be attempted, the variety prescribed depending, of course, on the group of muscles which you desire to have exercised. It is in such cases that the various forms of Zander's apparatus are so useful. But in all these voluntary exercises very great care should be taken not to fatigue the patient.

Contracture

In all severe cases of neuritis and in poliomyelitis you have to guard against the occurrence of contracture, and to the importance of this we have just alluded. In the so-called rheumatic facial paralysis it is very apt to show itself. If it does so, all electrical treatment should at once be given up and massage substituted. The affected muscles should be rubbed between fingers placed inside the mouth and outside on the cheek. The use of a wooden ball held inside the mouth, over which the muscles may be rubbed, is recommended by some. In cases of multiple neuritis it is very important that the feet be not kept continuously in an extended position. The pressure of the bed-clothes, which favours this, should be removed by means of a cradle, and the feet should be kept at right angles to the legs by a properly adapted sandbag or by light splints. Twice a day thorough

passive movements of the ankle-joint should be carried out, and, if there is any tendency to contracture at the knee, continuous extension by weights may be required. In poliomyelitis the same precautions have to be taken if the foot is affected, and the child should not be allowed to lie for long with the knee-joints flexed.

Surgical measures. In relation to paralysis from lesion of the lower motor neurone one or two things remain to be said. These, it is true, lie more within the purview of the surgeon than of the physician. But since the decision as to whether operative interference is likely to be useful and, if so, what measures are necessary, must rest with the physician, we may consider the question here. In doing so it will probably be best to take as our example the results of acute anterior polio-myelitis, as cases of this kind are more likely to be encountered than those other conditions which necessitate similar interference.

Tendon trans-plantation. Of all these measures the most important and useful is that known as tendon-transplantation. The operation consists essentially in replacing the action of a paralysed muscle by that of a healthy one. The tendon of the latter, either in part or in whole, is attached to the tendon

of the paralysed muscle, and in this way the sound muscle replaces, to some extent at least, that which has been lost. So far as I know, this method was first described in 1881 by Nicoladoni,[1] who in the following year successfully operated on a case of pes calcaneus, attaching the peronei to the stump of the tendo achillis.[2] Since that date the operation of tendon-transplantation has been practised by many, with generally satisfactory results. The method admits of many variations, according to the particular anatomical relations of the muscles involved. Lange,[3] for example, has succeeded in adding to the length of the tendon, where that was necessary, by attaching silk threads. In this way he united the cut ends of the biceps of semitendinosus to the tibia by an artificial tendon of silk, and so replaced the paralysed quadriceps. The variations in these operations are, as you may readily conceive, very numerous. A good description of the technique of these is given by Tubby and Jones,[4] to whose excellent work I may refer you for details.

Variations in the method.

[1] Nicoladoni, "Ueber den Pes calcaneus," *Arch. f. klin. Chir.* Bd. 26 (1881), S. 467.

[2] *Wiener med. Presse* (1882), S. 329.

[3] Lange, "Weitere Erfahrungen über seidene Sehnen," *Münch. med. Woch.* (1902), S. 10.

[4] Loc. cit.

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Let us suppose that a case of acute poliomyelitis of old standing comes to be dealt with. What is the duty of the physician? Clearly the first thing to do is to study the individual case carefully: to find out what particular movements are defective—what individual muscles are paralysed. Sometimes all the muscles acting on a joint are completely paralysed, and then there can, of course, be no question of tendon-transplantation, though it may be for the advantage of the patient to produce a more or less complete ankylosis. But as a rule this is not the case, and you will find that the paralysis is limited to a group of muscles or to certain individual muscles of a group. You will test the electrical reactions of all the muscles concerned very carefully, ascertaining which remain sound, which are completely paralysed, and which have potentiality of recovery. In deciding what it is possible for the surgeon to do so as to utilise what is left of muscular power and to restore the lost balance of force at the joint in question, you have to exercise a careful judgment so as to regulate the amount of power you are to have transferred from, say, a flexor action to an extensor. In choosing the muscle which is to

Duty of physician.

Choice of reinforcing muscle.

be utilised to reinforce that which has been paralysed, certain general principles ought to be followed. It is clear, for example, that the most satisfactory result will be obtained from a muscle which normally pulls in a line and direction parallel to the pull of the muscle which has become paralysed. Further, it is well, in most cases, to employ a member of the same muscle group, if such can be found in a healthy condition. Obviously, for physiological reasons, such an arrangement renders the subsequent movements of the joint more easy for the patient to learn. On the other hand, if you are compelled to use, say, a flexor muscle to reinforce a paralysed extensor, the patient, after recovery from the operation, is obliged to learn the new movement very carefully. This he has to do by the vicarious aid of the neurones of sight, very much in the way I shall have to describe fully when dealing with the treatment of tabes. No doubt the difficulties of the patient are thereby increased. But he usually overcomes these difficulties, and in some cases it is even of advantage to use a muscle of a group of antagonists, because in that way you lessen the opposition and allow the balance of power to be

Which are to be preferred.

Re-education of patient.

more readily restored. In no case of tendon-transplantation can the normal joint-power be fully restored, but very often a useless and troublesome limb may be made distinctly useful in the daily life of the patient.

Nerve anastomosis by operation.

Another surgical measure, the propriety of which we have occasionally to consider, is that of suturing a healthy motor nerve trunk to one which has become paralysed, and, by this nerve anastomosis, restoring function in the muscles which have become paralysed. This operation has been performed several times of late years in connection with facial paralysis,[1] the nerve selected for anastomosis being either the spinal accessory or the hypoglossal, the latter by preference. The results seem, on the whole, encouraging.

[1] See paper by Ballance and Purves Stewart, *British Medical Journal*, 1903.

LECTURE IV

ON THE TREATMENT OF PARALYSIS RESULTING FROM LESION OF THE UPPER MOTOR NEURONE

The upper motor neurone—Its degenerations—Primary lateral sclerosis—Symptoms of paralysis of this neurone—Infantile cerebral paralysis—Myelitis—Ataxic paraplegia—Disseminated sclerosis—Cerebral hæmorrhage.

Arrangement of the upper motor neurone.

THE cells of the upper motor neurone, with their protoplasmic processes or dendrites, lie, as you know, in the motor area of the cerebral cortex. The axones or axis-cylinders of these cells pass down the motor tracts, traversing in turn the internal capsule, the cerebral peduncles, the pons, the medulla oblongata (where the majority cross in the decussation), and the pyramidal tracts of the spinal cord—some, however, being diverted to the motor cerebral nuclei. They end in arborisations round the cells of the lower neurone in the anterior horns of the cord, and in the corresponding nuclei of the motor cranial nerves.

Course.

Through this long course the upper motor

neurone is exposed to many forms of injury. It may be attacked as a whole by the toxines of disease, but more usually the lesion which affects it is somewhat localised. The cells in the motor cortex may be destroyed by meningitic processes, by polioencephalitis, by cortical hæmorrhage, by thrombosis or embolism with subsequent softening, by the pressure of tumours, and by other like causes. In the neighbourhood of the internal capsule, the seat of election of cerebral hæmorrhage, the fibres of the motor tract,—that is, the axones of the upper motor neurone,—may be torn and compressed by such bleeding. Or hæmorrhages or tumours at lower levels, in or in the near neighbourhood of the pons or medulla, may seriously implicate the upper motor neurone. Similarly, as the axones stretch their long way down the cord they may be affected by local processes there. The pressure of broken, dislocated or diseased vertebræ, spinal meningitis, myelitis, tumours, hæmorrhages, may, one and all, produce paralysis by involving the upper neurone. And, in both brain and cord, disseminated sclerosis may cut the pyramidal tracts here or there with resulting paralysis.

And, just as in the case of the lower neurone,

Degeneration.

the trophic influences which determine the nutrition of the whole unit proceed from the cell, so in the case of the upper. Any disease which destroys the cell in the cortex causes the neurone as a whole to degenerate, and thus we have a descending degeneration of the axones of these cells—that is, of the motor or pyramidal tract in the brain, medulla and cord. Any one of the lesions just mentioned which cuts the axone causes the peripheral part of that fibre to degenerate, because it is thereby separated from its nutritional centre, the cell in the motor cortex.

System diseases.

As to the so-called system diseases of the cord in which the upper neurone is affected, such as primary lateral sclerosis, amyotrophic lateral sclerosis, ataxic paraplegia, I think that you will understand their position and etiology best if you consider them not as cord lesions but as lesions of the upper neurone. A toxine may so deeply affect the whole neurone as to cause degeneration of the cell with its dendrites, the axone and its arborisations—the whole unit in fact. If such a process go to the length of complete degeneration, traces of such degeneration would be found after death in the cortex, in the internal capsule, in the peduncles, pons, medulla,

and down the whole length of the cord. But in some cases the process does not, at first at any rate, go to this length. If you have merely so much disturbance of the functions of the cell as will lead to an impairment of its trophic influence, of its nutritional power, then that part of the neurone which is most difficult to nourish—that is, the part furthest removed from the cell—will suffer most and will first show signs of degeneration. Hence it is that in some cases the degeneration is found only in the cord, and often low down the cord, in the furthest extremities of the upper neurone. In this way you may explain to yourselves the method of origin of those cases of disease in the cord, in which the pyramidal tracts there are affected without there being any trace of visible degeneration in the parts of the upper neurone situated within the brain.

Toxic lesions. Either the severity of the poison was not sufficient to destroy the neurone as a whole, or sufficient time was not given it to develop its full effect before the patient died. It produced only so much depression of vital action in the cortical cell as to lead to degeneration of the more distant part of the axone—that is, degeneration of the motor strands in the cord.

In all likelihood, however, certain of the degenerations seen in the pyramidal tracts (and indeed some of those met with in other tracts also) are not to be considered as toxine lesions. They may, in some cases at all events, be regarded as the result of a slow decay of the neurones concerned, from exhaustion of vitality, rather than as a toxic manifestation. You know from your general experience and observation that many men show degenerative changes in their blood-vessels before they have reached that period of life at which such changes are to be expected. A similar early senility may be met with in connection with neuronic systems, and in some cases it seems likely that motor tract degenerations are of this nature. This hypothesis has been discussed by Mott and Tredgold in a very interesting way.[1]

Early senile changes.

There seems to be so much confusion regarding the disease known as primary lateral sclerosis, that I am tempted to enter into some short discussion of the subject here. The group of symptoms which form the clinical picture of

Primary lateral sclerosis.

[1] Mott and Tredgold, "Some observations on primary degeneration of the Motor Tract," *Brain* (1902), p. 401.

spastic paralysis was first described by Charcot in the year 1874,[1] and almost immediately thereafter by Erb.[2] These consist of a slowly advancing paralysis or paresis, chiefly of the legs, accompanied by stiffness and rigidity of the muscles, by marked exaggeration of the deep reflexes and usually by Babinski's sign. No form of sensation is affected, there are no vaso-motor or trophic changes, no genito-urinary or rectal troubles.

Charcot suggested, with great acuteness, that this group of symptoms must be due to lesion of the pyramidal tracts—an opinion which Erb shared; but neither of these great clinicians had had at that time an opportunity of examining the cord from a pure case of this description. The disease is a very chronic one and does not threaten life, and death is usually due to some intercurrent affection. As a rule the lesions found are complicated, and do not correspond to the theory of pure lateral sclerosis. Many indeed deny that such a disease exists.

Recorded cases.

The recorded cases which have, in their morbid anatomy, proved to be nearly pure

[1] Charcot, "Leçons cliniques sur les Maladies du Système Nerveux," sér. ii. fasc. 3. Paris, 1874.

[2] Erb, *Berliner klin. Wochenschr.* July 1875; and in *Virch. Arch.* Bd. 70, Heft 2, June 1877.

examples of primary lateral sclerosis are very few in number. The first is that of Morgan and Dreschfeld,[1] in which there was distinct degeneration of the pyramidal tracts in the cord extending up to the medulla, and apparently no other lesion. But, when the specimens were more completely examined, Dreschfeld[2] found that there was also some degeneration in the cells of the anterior horns. Strümpell's case[3] was likewise a nearly pure example of lateral sclerosis, but in it also there was some degeneration of the cells of the lower motor neurone, so that it also must be considered as a case of amyotrophic lateral sclerosis. In the year 1896 Dejerine and Sottas[4] published the case of an alcoholic who had, during life, exhibited symptoms of spastic paralysis. The pyramidal tracts were degenerated from the upper end of the cord downwards, but there was also some degeneration in the posterior columns.

[1] Morgan and Dreschfeld, "Idiopathic lateral sclerosis," *British Medical Journal*, Jan. 1881, p. 152.

[2] Dreschfeld, *Journal of Anatomy and Physiology*, vol. xv. (1881) p. 510.

[3] Strümpell, "Ueber einen Fall . . . mit den Symptomen einer allgemeinen spastischen Lähmung," *D. Zeit. f. Nervenheilk.* Bd. 5, May 1894.

[4] Dejerine and Sottas, "Sur un cas de paraplégie spasmodique," etc., *Arch. de Physiologie normale et pathologique*, vol. viii. (1896) p. 630.

A more recent case is that recorded by Ida Democh[1] from Hitzig's clinique in Halle, in which the symptoms during life were those of typical spastic paralysis in an alcoholic. After death there was found degeneration of the pyramidal tracts in the cord, but there was also slight degeneration of the columns of Goll, and a hydromyelia towards the lower end of the cord. Probably this case approaches as nearly as any yet met with to the pathological findings which ought theoretically to characterise primary lateral sclerosis. The condition of hydromyelia probably did not give rise to any symptoms, and the slight degeneration noted in the columns of Goll is not uncommon in chronic alcoholics. Erb in a recent paper[2] cites a few other cases of this kind, making eleven in all.

Diagnosis. One hears sometimes the diagnosis of primary lateral sclerosis made during life, but probably in almost every case this diagnosis is wrong. Pure instances of the affection must be of exceeding rarity. In truth the clinical picture of spastic paralysis, which is a very well defined one, may

[1] Ida Democh, "Ein Beitrag zur Lehre von der spastischen Spinalparalyse," *Arch. f. Psychiatrie und Nervenkrankh.* Bd. 33, 1900.
[2] Erb, "Ueber die spastische und die syphilitische Spinalparalyse," etc. *D. Zeit. f. Nervenheilkunde*, Bd. 23 (1903), S. 347.

be encountered at the early stage of several different diseases, among which may be mentioned amyotrophic lateral sclerosis, ataxic paraplegia, myelitis, syringomyelia, disseminated sclerosis, and neoplasm of the cord. In the majority of such cases the diagnostic signs of one of these lesions will appear subsequently. Very similar symptoms may also occur in hysteria.

Clinical signs of degeneration of the upper motor neurone.

You will remember that in speaking of the lower motor neurone it was pointed out that whatever part of that neurone was affected the resulting phenomena were essentially the same. A like rule holds good as regards the paralytic symptoms produced by lesion of the upper neurone. Whether the neurone is attacked as a whole or whether the axones are involved at some particular point in their course down the motor tract, the essential signs as regards paralysis are the same. These signs are, briefly, as follows :—

The muscles involved are withdrawn from the influence of Will; they are paralysed. But, in contrast to the condition which obtains in the case of the lower neurone, the paralysis here is spastic, the tonus of the muscles is increased, the deep reflexes are exaggerated, Babinski's sign

shows itself, clonus is often present, and there is a strong tendency to contracture. On these, taken together with the history of the case, the diagnosis is based, and without going further into the matter, it may be said generally that there is seldom any great difficulty in determining whether or not the upper motor neurone is involved in the lesion. What the seat of the lesion may be, and of what nature it is, are points which can only be settled by careful consideration of the symptoms as a whole, their order and mode of onset, the age of the patient, the condition of other organs, etc. The question of diagnosis does not, however, concern us here.

Treatment. As regards treatment it may be said, speaking generally, that lesions of the upper motor neurone are less amenable to treatment than are those of the lower. The upper neurone is of later development than the lower, more specialised, more delicate, more susceptible of injury and less capable of repair. Still, in many cases, treatment does great good.

Removal of cause. If the cause of the lesion can be determined, you will, of course, seek as far as may be to remove it. This point has been fully considered in

a former lecture so far as relates to the toxines of disease, to alcohol and to metallic and other poisons. In this connection you will recall the case of a man whom some of you saw, who was suffering from the results of chronic lead-poisoning, the poison having attacked the upper motor neurone. When we saw him for the second time, after he had been under treatment for some months with a view to removal of the lead salts from his tissues, we found that his spastic symptoms had disappeared.

Toxines.

The toxines of influenza, rheumatism, tubercle and syphilis, and of many other infective complaints are apt, especially in neurotic subjects, to attack the upper neurone. These demand in each case appropriate treatment. I have, for example, seen symptoms from the side of the upper motor neurone disappear after the removal of a tubercular testicle. And if you recall the nature of the lesion in many cases of infantile cerebral paralysis, that will remind you that the upper motor neurone may be attacked by toxine processes of unknown origin, and probably there are many varieties of these.

Infantile cerebral paralysis.

The treatment of those cases of infantile cerebral paralysis which arise from the action of

some toxine on the motor cerebral cortex, is no more satisfactory than that of the corresponding lesion of the anterior horns of the spinal cord. The patient as a rule comes under observation only after the cortical destruction has gone its full length. The paralysis, which usually affects one side of the face, one arm and one leg, but chiefly the arm, tends to improve somewhat in the months which follow the occurrence of the lesion, and this improvement may be aided by massage. Beyond using cod-liver oil and tonics (among which strychnine should not be included in the cases in question, at least in the early stage) medicinal treatment is of little avail.

Use of practice. After the acute stage has completely passed away, I have seen some good follow the practice of tying up the healthy arm so as to force the child in his play to practise moving the affected hand. In this way he gets back some of the power which he had lost, and it is not too much to imagine that fresh centres may thus to some extent be educated to take on the work of those which the toxine has destroyed. Carefully devised gymnastics will also do good, but they must not be so protracted or so energetic as to produce any considerable fatigue.

As medicine can do so little, may we look to surgery to help us in these very hopeless cases? Certainly in many instances of the cerebral hemiplegia and diplegia of infancy, a little can be done by the surgeon to make life more tolerable and easy. I do not here deal with the question of the epileptic attacks which so often show themselves. These will be considered in the succeeding lecture. But, apart from this, in some cases of infantile cerebral hemiplegia, where there is thickening of the membranes and scarring of the cortex, the operation of trephining may do good. I have seen some little benefit follow this procedure in one or two cases. Much improvement can hardly be expected.

Surgical treatment.

Trephining.

In regard to the limbs there are also possibilities of considerable improvement from surgical interference. Indeed, in many cases, operation may render a useless limb tolerably useful. The principles on which operative interference rests are very much the same as in acute anterior poliomyelitis, and these I have sufficiently detailed in the last lecture. In the spastic conditions with which we are now dealing, the contracture of certain muscles causes such stretching and elongation of their

Relief of spastic condition.

antagonists that these latter muscles are placed under conditions which render their nutrition difficult and their restoration to normal function impossible. The surgeon can relieve the spastic contraction by careful tenotomy, and after this has been performed the antagonists may be restored to function by means of massage and faradism. In Little's disease, for example, you remember how firmly the muscles of the calf are contracted, so much so indeed that the child walks on tip-toe, being unable to bring the heel to the ground. The extensors are thus very much stretched and elongated. If, now, the tendo achillis be divided and the foot brought to a right angle with the leg, these extensors are relieved, and, by massage and faradism, they may be brought back to a fair strength and vigour. In a similar fashion the contractures which determine the extreme adduction we so often see, may be dealt with. For details as to the procedure and as to the possibilities and limitations of tendon-transplantation in these cases, I refer you to the useful book by Tubby and Jones[1] which I have already mentioned. Of course, only certain cases are suitable for

Tenotomy.

Division of tendo achillis.

Tendon-transplantation.

[1] *Modern Methods in the Surgery of Paralysis.* London, 1903.

these operations on the limbs. They should not be attempted in cases in which there are athetotic movements, or frequent fits, nor where the patient's mental capacity is so low that he is unable to learn and practise the movements necessary to be taught after such operations. *Contra-indications.*

Some further remarks as to the subsequent treatment of these cases will be made towards the end of this lecture, when we come to consider the after-treatment of spastic paralysis generally.

Myelitis.

Myelitis, another disease in which the upper motor neurone suffers, its axones in the cord being involved in the inflammation, is also in almost every case, if not invariably, the result of the action of micro-organisms, or their toxines. Syphilis is very commonly the cause, and in such cases the myelitis usually comes on within two years of the date on which the primary sore showed itself. Singer,[1] for example, notes that out of nineteen consecutive cases, fifteen had recently had syphilis and one had probably suffered from that malady. This quite coincides with the results of my own observation. *Causation of.*

[1] H. D. Singer, "The pathlogy of so-called acute myelitis," *Brain*, vol. 25 (1902), p. 332.

When the myelitic symptoms appear in a person the subject of syphilis a vigorous course of antisyphilitic treatment should be commenced at once, and it is necessary to push the administration of mercury as rapidly as possible. Hypodermic administration of the drug finds its proper use in acute cases of this kind, as indeed I have told you in a former lecture.

Serum treatment.

Apart from syphilis, the cord may be invaded and myelitis started by various unkown toxines. Among the micro-organisms of known character which have been proved to act in this way may be mentioned the following:—Streptococcus, staphylococcus, pneumococcus, influenza bacillus, bacillus coli communis. This microbic origin of myelitis would suggest that a serum treatment is that likely to be of most avail in such conditions. There is, however, only a small number of such serums available, and even those which are procurable have shown themselves to be useless in these cases. Even in that myelitis which can be produced in animals by inoculation of the spinal meninges with cultures of known organisms, and therefore under conditions in which serum therapeutics can be most favourably carried out, no curative result has been

attained. Marinesco,[1] for example, experimenting with streptococcus in this way, found that even when the culture and the anti-streptococcus serum were given together (he used Marmoreck's serum) no very evident effect was produced, save perhaps that the animals so treated did not die quite so rapidly as the control animals did. The future will, however, almost certainly place in our hands the means of carrying out satisfactory serum treatment in cases of myelitis.

Rest and other general measures.

But, while awaiting and expecting the advent of a series of satisfactory serums, we must in the meanwhile continue to treat cases of myelitis on the old and empirical lines. Absolute rest in bed is of course essential, and the patient should not lie on his back. This would make the spine the most dependent part of the body—an undesirable position for an inflamed organ. He should rest either in the prone posture or on one side or the other Certainly, in all acute cases of this description, the patient should be placed at once on a water-bed. This may avoid, and will in any case lessen, future trouble from trophic disturbances—bed-sores and the like.

[1] Marinesco, "Nature et traitement de la myélite aigue," *Nouv. Iconographie de la Salpêtrière*, November to December 1900.

At the commencement of an acute myelitis some good may result from giving the patient a vapour bath, but this should be done in bed and without any unnecessary movement. The bowels are to be freely opened by means of a dose of calomel, and some observers have seen benefit follow the application of leeches over the spine. The method of lavage of the tissues by means of rectal irrigation, previously described (p. 49), may be tried in acute myelitis in the hope of washing away some part at least of the toxines which are being formed.

The diet during the acute stage should be that of fevers generally. As to medicines, it is usual to give mercury in some form, and salicylate of sodium may occasionally be of benefit. Ergot has been given in these cases, but I have never seen any benefit follow its use. In the later stages iodide of potassium should be used. But the medicinal treatment of myelitis is very unsatisfactory and disheartening.

Attention to bladder.

Attention to the condition of the bladder is of the highest importance in all cases of myelitis. If there be retention of urine, a catheter ought to be passed, and the bladder thoroughly emptied. It is likely that the instrument will require to be

used very frequently, and the most scrupulous care must be taken to avoid the introduction of organisms. It should be remembered that in cases of myelitis the mucous membrane of the urinary tract has much less vitality and resisting power than in the normal condition, and is therefore more easily infected during the operation of passing a catheter. If cystitis does occur, *Cystitis.* and even in most careful hands this complication sometimes arises, the bladder should be washed out at least twice in each twenty-four hours with some antiseptic solution. Boracic lotion (gr. 10 to the ounce) does very well, or solution of quinine; or very diluted perchloride of mercury solution may be used. But in these cases you ought always to give antiseptics by the mouth at the same time. I have usually employed boracic acid or salol for this purpose. These appear to give the best results, but I have once or twice seen marked benefit follow the administration of urotropine. This substance, which is more correctly designated as hexamethylene-tetramine, is believed when it reaches the blood to break up into ammonia and formaldehyde. The latter, which is of course a strong antiseptic, passes into the urine.

Bed-sores. Another very important point is, by careful nursing, to avoid the formation of bed-sores, from which indeed many patients die. The readiness with which such sores form on the sacral region and at other parts where pressure comes, is to be explained partly on the ground of the low vitality of the skin and partly by reason of the fact that the patient cannot feel that the pressure has at any time been too long continued, and cannot turn so as to relieve that pressure. The skin also is moist with decomposing perspiration, and is apt to be soiled with urine and fæces, especially if the evacuations are involuntary. The nurse who is careful to avoid this distressing complication will see that her patient is placed upon a water-bed, and that the bed linen and the night-dress of the patient are frequently changed. She will see that he is turned from time to time from one side to the other. She will wash the skin of the back at least twice a day, and, after thoroughly drying it, will rub in some strong spirit, dusting it over, finally, with boracic acid powder. If necrosis of the skin does take place, and this will sometimes happen even with the best nursing, the slough should be kept dusted with iodoform until it separates.

After this the sore is best dressed with boracic lotion.

It may be well, at this point, to remind you that disease of the upper motor neurone does not of itself give rise to vaso-motor, or trophic symptoms, or to incontinence of urine and fæces. It is only when, as in myelitis, the lesion passes to other parts of the central nervous structures that we see added trophic changes in the skin, muscles, joints, etc., vaso-motor disturbances, and bladder and rectal troubles.

Pain.

Pain in acute myelitis usually yields to the action of phenacetin. In such cases it is probably best to give that drug in combination with salicylate of sodium and sulphate of quinine. When the pain is not stilled by these remedies, it may be necessary to give morphia. Another very distressing symptom is that of twitching and jerking of the legs. Bromide of potassium usually helps this, and sometimes atropine is of service. If the patient is in a state to allow of his being lifted from bed and placed in a warm bath he will experience great relief. If the jerking is severe and incessant, the administration of morphia may become necessary.

Muscular twitching.

When the disease passes out of the acute

Chronic myelitis. into the chronic stage the possibilities of favourably influencing its progress do not increase. Occasional courses of iodide of potassium are indicated with the object of promoting phagocytic action and of removing the exudation. A certain amount of good may be accomplished by means of massage, and in the case of such muscles as still possess some possibility of voluntary movement faradism may be advisable. Otherwise all we can do is to maintain the nutrition by giving plenty of light food and by general tonic treatment. The patient should be encouraged to spend as much time in the open air as he can. Graduated exercises are of some service, but their potentiality for good is very much less in cases of lesion of the pyramidal tracts than it is where, as in tabes, the lesion is one affecting the centripetal co-ordinating apparatus. The reason of this is clear. In the latter case there are, as will be explained later, alternative neurones which may be brought into vicarious use. In lesions of the pyramidal tracts no alternative paths are at the disposal of the organism.

Ataxic paraplegia. In ataxic paraplegia, in which degeneration of the centripetal neurones conveying muscular sense is added to that of the pyramidal tracts,

the evidence of toxic action is very strong. Consequently, in this malady, especially if the case be seen at a comparatively early stage, efforts to obtain the elimination of toxines should be strenuously made on the lines detailed in Lecture II. In the later stages of the affection some benefit may result from those graduated exercises which will be described when we come to consider the treatment of locomotor ataxia.

Disseminated sclerosis.

Disseminated or multiple sclerosis, like myelitis, has in all probability a micro-organismal origin in most cases, but it may also occur as the result of the action of certain metallic poisons such as lead. Alcoholism also is one of its causes. As the patches of sclerosis involve the axones of the upper motor neurone at some point or another, and usually at many points, a word may be said here as to its treatment. Clearly if you can find evidence that lead or some similar metal is acting, your line of treatment will be evident. The same may be said of those cases in which syphilitic infection has preceded the attack.

Removal of cause.

But in those instances in which there is no evidence of such causes, the treatment is not in any sense hopeful or satisfactory. Of the drugs

Drugs.

used, that which seems to do most good is nitrate of silver, and I have certainly seen benefit follow its administration. Prolonged courses of arsenic, in increasing doses, are said sometimes to influence the disease favourably. Müller, in a recent work[1] on multiple sclerosis, states that he has used with some benefit a combination of arsenic, strychnine, quinine, and iron. A suitable pill of this kind may be prescribed as follows :—

> ℞ Acidi arsen., gr. $\frac{1}{130}$-$\frac{1}{60}$
> Extr. nucis vom., gr. $\frac{1}{12}$-$\frac{1}{6}$
> Quin. hydrochl.
> Ferri lact. āā gr. $\frac{3}{4}$
> Ext. gent., *q.s.*

I have lately seen distinct improvement follow the use of static electricity in early cases of multiple sclerosis. But in connection with all these forms of treatment it has to be remembered that the disease is apt to show periodic remissions, and that what improvement takes place may not be due to treatment.

The patient suffering from multiple sclerosis should be advised to lead as easy and equable a life as may be possible to him, to maintain his nutrition at a high level, to winter in a mild

[1] Müller, *Die multiple Sklerose des Gehirns und Rückenmarks.* Jena, 1904.

climate if he can, and to avoid strain of all kinds. Excesses *in venere* are specially harmful, and alcohol should be sparingly used; indeed, having regard to the sensitiveness of the upper neurone to this drug, the patient would be safer if he abstained from it use.

General measures.

Gowers has shown that pregnancy and labour are apt to accelerate the progress of disseminated sclerosis, and female patients should, when necessary, be informed of this risk.

Results of pressure on upper motor neurone.

Let us turn now to the more mechanical side of the question and consider the results of pressure on the upper motor neurone. It is to be remembered that the whole length of that neurone, in the adult at any rate, lies in a cavity surrounded by bony walls. It differs essentially in this respect from the lower neurone, the greater part of which is situated in the soft tissues. Within the skull or the spinal canal any tumour, or any effusion of blood or serum, may give rise to pressure sufficient to compress the upper motor neurone and interfere with its functions.

Sudden increase of intracranial pressure.

The effects of increased intracranial pressure are strikingly seen in cases in which that pressure arises suddenly. The simplest example of

Extra-dural hæmorrhage. this is that extra-dural hæmorrhage which follows the rupture of the middle meningeal artery—a condition usually, though not always, the result of trauma. There can be no doubt here as to what the treatment should be, and some of the most brilliant results of modern cranial surgery have been those which followed trephining and ligature of the vessel in such cases.

Cerebral hæmorrhage. The matter stands otherwise when we come to consider the question of ordinary cerebral hæmorrhage—say into the lenticular nucleus and neighbouring parts. Here the indications for treatment are not so clear and the results of that treatment are not nearly so satisfactory as in the case we have just referred to. The hæmorrhage tears up and destroys more or less completely the parts into which it first breaks, and, among them, axones of the upper motor neurone in the internal capsule are usually to some considerable extent included. *Mechanical results of.* The mechanical shock of the escaping blood traverses the brain substance in all directions like a wave, until it breaks on the cranial bones. If the effused blood be of any amount, it produces an increase of intracranial pressure, to which indeed the compressed and flattened con-

volutions, often seen after death, strikingly testify. In this way such fluid as can be made to leave the cranium—blood, lymph, cerebro-spinal fluid—is forced out, and considerable anæmia (especially of the cortex) is brought about. In the immediate neighbourhood of the hæmorrhage the venous return is apt to be impeded with resulting œdematous swelling. This still further increases the pressure on more outlying parts.

Coma.

The coma which follows the apoplectic attack is the outward expression of the cortical anæmia caused in such ways as the above, and perhaps also produced reflexly from the irritation of hæmorrhage in the central grey matter.

Treatment of apoplectic attack.

If, now, you are called to see a case of apoplexy from cerebral hæmorrhage in the stage of coma, what are the indications for treatment? It is clear, in the first place, that any hæmorrhage which is still going on should be arrested. No unnecessary movement of the patient should take place. He should be gently conveyed to the nearest bed and allowed to lie there in such a position and with his clothing so arranged as to allow of the free return of venous blood from the

head and neck. If the diagnosis is certain and if there are signs of a continuing hæmorrhage. and if the site of the lesion is the usual one, it may be advisable to compress the carotid artery in order to diminish the rate of the circulation and lower the blood-pressure, and so allow of clotting and the arrest of hæmorrhage. It may even be advisable to ligature that vessel in special cases, as in one recorded by Dercum and Keen,[1] where this procedure produced very satisfactory results. I cannot, however, speak from experience with regard to the results of this operation, never having seen my way clear to recommend its performance. Perhaps the administration of chloride of calcium would accelerate the clotting of the blood in these cases. In animals at any rate it appears to have a marked effect in this direction.

Compression of carotid artery.

It has been pointed out that the cause of the coma in such cases is in all probability anæmia of the cortex resulting from increased intracranial pressure. This pressure not only causes coma but also injures other parts of the brain which have not been directly destroyed by the hæmorrhage. The blood-pressure and consequently

[1] Dercum and Keen, "Two cases of Ingravescent Cerebral Hæmorrhage treated by ligation of the common carotid artery," *Journal of Nervous and Mental Disease*, vol. xix., (1894) p. 586.

the intracranial tension can be considerably reduced by free purgation. For this purpose croton oil or calomel may be usefully employed, and the lower bowel may be emptied by means of an enema. *Purgation.*

By far the quickest and most effectual way of reducing the intracranial tension is by blood-letting. Venesection, formerly so much employed, has fallen into an unreasonable disrepute. He who is bold enough to abstract blood in suitable cases may see the coma shortened and the general symptoms ameliorated. But before proceeding to abstract blood either by opening a vein or by the less objectionable method of applying leeches to the neck, he must first of all make a clear and certain diagnosis of cerebral hæmorrhage, for such a practice as blood-letting in the case of embolism or thrombosis would be very harmful. The cases in which venesection is likely to do good are those in which the face is darkly flushed, the pulse slow and full and the coma deep and of long duration. The good effects of venesection in suitable cases depend not merely on the removal of a certain quantity of blood from the circulation, but also on the fact that this reduces cyanosis. One of the results of *Blood-letting.*

dyspnœa is contraction of the arterioles and a consequent high-blood-pressure, and the reduction of the cyanosis lowers the blood-pressure materially.

Some effect towards lowering the blood-pressure may also be produced by the application of heat to the extremities and the trunk. The dilatation of the cutaneous vessels produced in this way will, to some extent, relieve the intra-cranial pressure. The action will be augmented by the application of an ice-bag to the head.

Attention to bladder.

During the continuance of the coma, and indeed for some time thereafter, the condition of the bladder should be carefully watched. There is often retention, calling for the use of the catheter; and sometimes there is incontinence of urine. In the latter condition great care is required on the part of the nurse to keep the skin clean, as bed-sores are liable to form and to give trouble.

Feeding.

As to the feeding of such cases there is sometimes difficulty. If the cardiac strength is good, there is no need to feed at all during the stage of coma. But if the pulse is weak, rectal feeding should be resorted to. In the days following the attack the food should be light and simple.

After the stage of coma has passed, the use of iodide of potassium will aid the absorption of the extravasated blood, and in that way help in restoring the function of those axones of the upper motor neurone which have not been actually destroyed by the lesion.

Absorption of extravasated blood.

The process by which blood extravasation in the brain is absorbed and removed is an interesting one. No doubt, as in the case of other forms of solid exudation, the removal is effected in part by the exudation becoming fluid and being thus absorbed. But certainly the most important rôle in the absorption is that played by the phagocytes. These phagocytes, in such cases, are for the most part polynuclear neutrophile leucocytes, though some may be altered endothelial and connective tissue cells. This phagocytic action is of great importance to the organism in these cases, because, as the extravasation is removed, the pressure on neighbouring parts of the brain substance is relieved; and if the pyramidal tract has not been directly torn up, but has only been compressed, a more or less complete recovery may be expected to take place.

Phagocytic action.

Therefore one of the main objects which we

should aim at in treating these cases, is the stimulation of phagocytosis. No doubt a full nutrition will aid this, and in the stage which follows that of reaction the diet ought to be as full and strengthening as the powers of digestion of the patient will allow. But the means which, above all others, is useful in this stage is the administration of iodide of potassium. I have already alluded to this, and have said that Heinz's[1] observations, though not to my mind quite conclusive, strongly indicate that the value of iodide of potassium in these and other similar cases lies in the fact that it promotes and heightens phagocytosis. This conclusion is rendered the more likely in that these experimental observations are confirmed by clinical evidence.

Iodide of potassium.

Theory of its action.

In the next lecture we shall consider what effect on the upper motor neurone will be produced by a slow and gradual rise of intracranial pressure, and how this is to be met, in treating such cases.

1 Heinz, "Ueber Jod und Jodverbindungen," Virchow's *Arch.* Bd. 155.

LECTURE V

ON THE TREAMENT OF PARALYSIS RESULTING FROM LESION OF THE UPPER MOTOR NEURONE (*continued*)

Chronic hydrocephalus—Intracranial tumours—Intracranial abscess—Hæmato-myelia—Tumours of the spinal cord—Tuberculous disease of the spine—Destruction of the neurone from softening—Cerebral thrombosis and embolism—Management of special symptoms common to the foregoing diseases.

Slow increase of intracranial pressure.

In the last lecture we considered the results of a sudden rise of intracranial pressure on the fibres of the upper motor neurone. In contrast with that rapid increase of intracranial tension there stands the slow but very steady and great increase seen in chronic hydrocephalus. In the large proportion of these cases the lesion is due to some obstruction of the drainage from the lateral ventricles into the sub-arachnoid space, whether that obstruction is in the iter, in the fourth ventricle, or at the foramen of Majendie or the other openings in the sub-arachnoid

Chronic hydrocephalus.

meshwork. The effect of this is that as the fluid continues to collect in the ventricles and finds no way of escape, the ventricular system becomes distended and the intracranial pressure gradually rises. As a result, the brain substance becomes atrophied, being compressed between the ventricular fluid and the walls of the cranial cavity. The atrophy is, I think, accelerated by the fact that as the intracranial pressure rises and becomes more and more nearly equal to the pressure at the highest part of the blood wave, less and less blood enters the brain and its nutrition seriously fails. In the course of this brain atrophy the upper motor neurone suffers, degeneration passes down the pyramidal tracts, and the limbs show signs of spastic paralysis.

Its results.

Treatment.

In such cases medicinal treatment is only of avail where the primary lesion is a syphilitic meningitis. Probably in all cases, and certainly when there is any evidence of syphilis, an energetic anti-syphilitic treatment should be given.

Removal of fluid.

Any effort to check the accumulation of fluid by application of plaster or bandages to the head, obviously wrong in theory, is worse than

useless in practice. It is not difficult to relieve the pressure temporarily by tapping one of the lateral ventricles. This may readily be done by inserting an aspirator needle through the lateral corner of the anterior fontanelle. The procedure requires to be repeated from time to time as the fluid re-accumulates, because it is not advisable to leave in a permanent drain. The operation is at best only palliative, and it is not to be recommended save in special cases—for example, when convulsions are occurring frequently.

Trephining.

When the obstruction lies, as it often does, at the foramen of Majendie, the operation described by Lees and Barlow[1] of trephining the occipital bone and so getting at and draining the fourth ventricle, is no doubt the best in theory. The results in their cases were not, however, satisfactory. If any operation is to be performed at all, it should probably be that described by Sutherland and Watson Cheyne,[2] whereby a permanent drain is established between one lateral ventricle and the sub-dural space.

Drainage.

[1] Lees and Barlow, in Clifford Allbutt's *System of Medicine*, vol. vii. p. 558.

[2] Sutherland and Watson Cheyne, "A case of Chronic Hydrocephalus treated by Intracranial Drainage," *Transactions of the Clinical Society*, 1898.

In a case recorded by Schramm[1] this operation was followed by remarkable improvement. He used a drain of formalin-catgut which had been soaked for twenty-four hours in a 1 per cent solution of chromic acid and then for twelve hours in absolute alcohol.

Lumbar puncture.

A consideration of the usual method of origin of these cases would lead one at once to assume that any attempt to drain the ventricular system by means of Quincke's lumbar puncture would be useless, and this assumption is justified by clinical experience. In very few of the cases in which it has been tried has any good resulted. The reason for this failure lies, of course, in the fact that, as the obstruction is situated between the ventricles and the spinal meninges, no drainage of the latter can be of avail.

Brain tumours.

Intracranial tumours, in the great majority of cases, affect the upper motor neurone. They do this either by injuring the motor area of the cortex or its sub-cortical fibres directly or indirectly by reason of the general increase of intracranial pressure which they produce. The

[1] Schramm, "Beitrag zur Behandlung des chronischen Hydrocephalus mittels intracranieller Drainage," quoted in the *Jahresber. über die Leist. u. Fortschr. der Neurologie, etc.*, vol. iii. p. 993.

pressure of the growing tumour leads to œdema of the neighbouring brain-substance, and then to a low form of inflammation and softening. Other varieties of tumour infiltrate, their elements becoming mingled with the cerebral tissues which they destroy as they grow. As the tumour increases in size, the intracranial pressure rises and occasions those signs which indicate that a neoplasm is present within the cranial cavity. The effects of such a tumour upon the pyramidal tracts are, as a rule, at first irritative and subsequently paralytic. It is only with the phase of paralysis that we have here to do. The results of irritation will engage our attention in the next lecture.

Mechanical effects.

A tumour involving some part of the motor area will usually at first give rise to Jacksonian epilepsy, but presently it will be noticed that there is some temporary paralysis after each attack. This probably results from exhaustion of the cortical cells which have been irritated, but as the tumour grows, these cells are thrown more or less completely out of function, and the paralysis becomes permanent. But while this is the usual order of events, cases are sometimes met with in which the paralysis comes

Jacksonian epilepsy.

Paresis.

Paralysis.

without preceding spasm, and a monoplegia is the first localising symptom of a tumour in the motor cortex. In any case the paralysis is limited, at any rate at first, and shows itself as a monoplegia of the face, of the arm, or of the leg. Or it may be even more localised than this, and may involve, for example, only the extensors of the great toe. There will be found in such a case, however, some slight paresis of other muscles of the limb whose cortical centres lie close to those of the muscles which are fully paralysed. As the tumour grows this paresis will deepen into full paralysis. The paralysis takes the type of that of the upper motor neurone, the muscles preserving their volume, showing a high tonus and giving exaggerated deep reflex responses and Babinski's sign. Occasionally there may be some muscular wasting, but there is never any reaction of degeneration.

Hemiplegia. Tumours in the central ganglia usually involve the pyramidal tract in the internal capsule, and so give rise to a hemiplegia. The fibres of the upper motor neurone are so closely grouped at this point that they usually suffer together, but there are a few cases on record in which

the encroachment of the tumour was so partial that a monoplegia was produced.

Crossed paralysis.

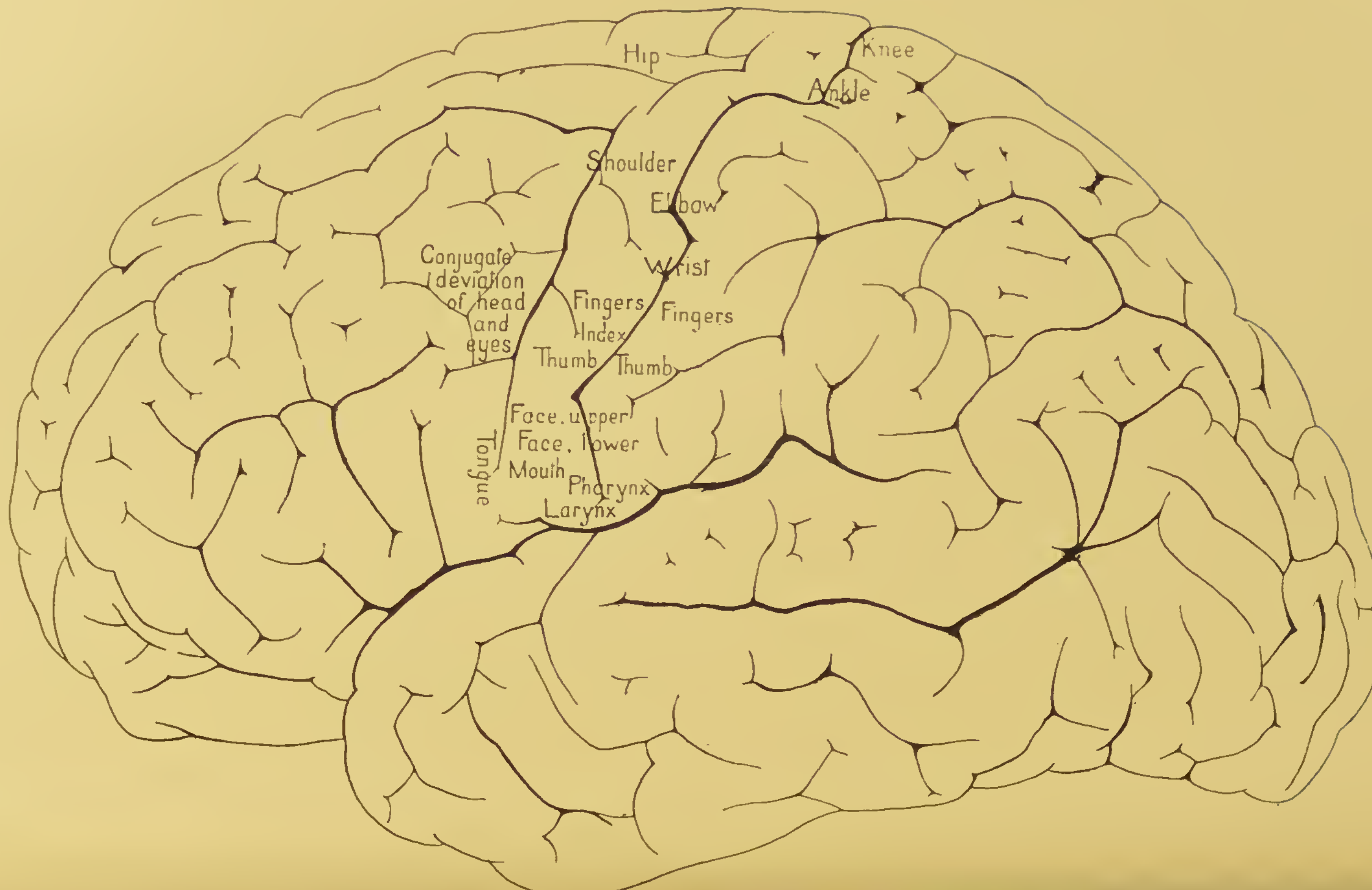
When the crus is involved, there results a hemiplegia with crossed paralysis of the oculomotor nerve, and when the tumour attacks the pons, the usual result is that the arm and leg on the opposite side of the body are paralysed, and the face on the same side. This is not, however, the place to enter into further detail with regard to these points.

Treatment of brain tumour.

The results of treatment in cases of brain tumour are far from satisfactory. Drug treatment is of avail, as a curative agent, only in a small percentage of cases, and surgery in a still smaller. I do not mean that brilliant results are not occasionally attained, but the number of cases which are permanently benefited forms but a very small percentage of the whole.

Antisyphilitic treatment.

Medicinal treatment is almost limited to cases of gumma—that is, in so far as actual cure is concerned. Palliative measures may be usefully employed in every case. In cases of brain tumour which, from the history of the case or for any other reason, are believed to be syphilitic, the most vigorous antisyphilitic treatment ought at



APPROXIMATE POSITION OF MOTOR AREAS ON LEFT CORTEX—*after* DEJERINE.

once to be commenced. Iodide of potassium has a wonderful effect in such cases, but to exert its full influence it has to be given in large doses. It is well to begin with 10 grains three times a day, and rapidly increase the quantity taken until at least 90 grains are taken in each twenty-four hours, and it is often advisable to go considerably beyond this quantity. In many cases it is necessary to order at the same time a course of mercurial inunction. Under these influences the symptoms of cerebral gumma abate, and in many cases disappear with a wonderful rapidity.

Iodide of potassium.

The advantages to be gained from the use of iodide of potassium are not absolutely limited to instances of syphilis, though they are much more conspicuous in the case of a gumma than in that of any other cranial tumour. Considerable improvement sometimes follows its use in cases of sarcomatous, gliomatous and tuberculous growths. Quite lately I saw the symptoms in a case of tuberculous tumour undergo a marked improvement as the result of the action of this drug.

Arsenic.

Arsenic is said to be of service in some cases of cranial tumour, but I have not seen any noteworthy benefit follow its use.

In cases of tubercle the administration of cod-

Treatment of tuberculous tumours.

liver oil and of tonics (particularly, I think, iodide of iron) is indicated. When the symptoms are not severe some improvement may be expected from open air treatment. This practice is certainly of benefit in cases of tuberculous disease of the spine.

Treatment of the headache of brain tumour.

Beyond these indications medicine is only of use in cases of brain tumour in combating symptoms. Of these the most severe and intractable is headache. Antipyrine and its allies may sometimes succeed in diminishing the pain, but usually (short of operation) the only means in our power is the use of morphia.

Surgery of brain tumour.

If, then, medicine can help but little in cases of intracranial tumour, how much assistance may we expect from surgery? The statistics regarding the operability of such tumours, contained in the works of Hale White,[1] Seydel,[2] Bramwell,[3] and Oppenheim,[4] and particularly the mass of facts contained in von Bergmann's work,[5] do not

[1] Hale White, *Guy's Hospital Reports*, vol. xliii. (1886), p. 117.

[2] Seydel, *Verh. der deutsch. Gesellschaft f. Chirurgie*, Bd. 21 (1892), p. 32.

[3] Byrom Bramwell, *Transactions of the Medico-Chirurgical Society of Edinburgh*, Session 1893-94.

[4] Oppenheim, *Die Geschwülste des Gehirns*. Wien, 1896.

[5] E. von Bergmann, *Die chirurgische Behandlung von Hirnkrankheiten*, 3te Aufl. 1899.

encourage optimism. As far as these statements go, it would appear that out of a hundred cases of brain tumour, about five are under conditions which justify the performance of an operation with a view to the removal of the growth. Probably about one-half of these operations are successful, but there is no certainty as to this point, as unsuccessful cases are not always published. At the commencement of the year 1903, Starr[1] was able to collect from various sources the records of 365 cases of brain tumour in which an operation for the removal of the growth had been attempted. In about 45 per cent the operation had been successful, the tumour having been removed and the patient having recovered. In many of these 365 patients the tumour was not found at the time of the operation, or, being found, was not so placed or of such a nature as to permit of removal. It is interesting to note that in those cases in which removal of the tumour was effected, the mortality was about 26 per cent.

Statistics of operations.

What are the points, then, which may guide us in deciding to call on a surgeon to operate in

Points determining operation.

[1] Starr, "The results of surgical treatment of brain tumours," *Journal of Nervous and Mental Disease*, vol. xxx. (1903), p. 398.

a ease of intraeranial tumour? It is clear, in the first plaee, that it is only after a thorough trial has been made of medication by means of iodide of potassium, and after that has been proved to be ineffeetual for removing symptoms, that there should be any appeal to surgery. Regarding this point a word may be added to what has been already said. It has been stated that in cases of gumma, especially when seen at a eomparatively early stage, treatment by means of iodide of potassium clears the symptoms away wonderfully quickly. There are, however, cases, and especially old standing cases, in which this satisfaetory result is not completely attained. In these an operation may subsequently beeome neeessary to remove scar tissue and thiekened membranes. A case of this kind eame under my treatment some months ago. The patient had aequired syphilis in South Ameriea many years ago, and had not then been subjected to proper treatment. The brain symptoms had existed for some time before he came under my notice, and although they yielded to a eonsiderable extent to treatment with iodide of potassium and mercurial inunction, they did not completely disappear. An operation was neeessary.

Another necessary condition preliminary to operation is that the site of the tumour should be localised. It is, of course, very desirable that this localising diagnosis should be perfectly exact, but I am far from holding that absolute certainty is necessary. If other things are favourable, a strong probability that the tumour in question lies at a particular point would justify interference, if the symptoms were severe and the need for operation pressing. There are, however, many regions of the brain from which a tumour cannot be removed, even with the advances of modern surgery. Tumours of the medulla, pons, base of the brain, basal ganglia, ventricles, corpus callosum, and those lying deep in the hemispheres, are all beyond the reach of operation. And it is only very exceptionally that a cerebellar tumour presents itself under sufficiently favourable circumstances (in one lateral lobe, and near its lower and posterior surface) to justify an attempt being made to remove it.

Localisation of tumour.

Therefore, you see, it almost comes to this, that only those tumours, cortical or sub-cortical, which lie on the convexity of the hemispheres, are capable of removal. The majority of those

Tumours suited for removal.

brain tumours which have been removed have been in the motor area, probably to a large extent because they are more easily recognised and localised when they are so situated. Of the long list of such tumours contained in von Bergmann's work, over 70 per cent lay in the motor area.

But, unfortunately, not by any means all of the tumours so situated are removable. The operation is the more easy to perform the nearer the tumour lies to the surface, the more definitely it is encapsulated, and the smaller it is in size. A large size is not, *per se*, a contra-indication to operation. Von Bramann, for example, removed a tumour weighing more than eight and a half ounces. But if a considerable portion of the brain tissue is infiltrated by the tumour growth, a satisfactory removal would be impossible.

Character of tumour.

The character of the tumour is therefore of great importance in this regard, but unfortunately in many cases a diagnosis of its nature is not possible. Carcinoma, which is almost always secondary to similar tumours elsewhere, does not lend itself to operation. Tubercular growths in the brain are often multiple, and in this case as well as when there is active tubercle else-

where, operation is not to be recommended. But when the tuberculous brain tumour is single and accessible, there is no reason why it should not be removed. Such tumours, however, lie very frequently in inaccessible situations in the cerebellum and about the base of the brain. Sarcomata, which are invariably surrounded by a capsule, are very favourable for removal, as are also fibromata and cysts. Gliomata are apt to infiltrate the brain substance, and this renders their removal difficult, but not always impossible. Allusion has already been made to the occasional necessity for recommending operation in cases of syphilitic gummata.

Palliative operation.

But, while surgical interference is of curative effect in but a very small proportion of cases of brain tumour, it is capable of affording relief in many. When the surgeon, having made a window in the cranium, and having laid the tumour bare, finds that its complete removal is not possible, he may greatly relieve the symptoms by excising a portion.

When the patient is suffering severe pain, or when blindness is increasing, a palliative operation is indicated even in cases in which the

position of the tumour cannot be localised, or, being localised, is known to be in some inaccessible region. In these circumstances the operation of trephining, by relieving intracranial tension, diminishes or may even remove many of the distressing symptoms to which that tension gives rise. In particular, the agonising pain, the vomiting, the convulsions, and the mental symptoms, if these exist, are relieved or removed by such an operation. The optic neuritis also, if it have not gone too far, may be checked and the further progress towards optic atrophy may be arrested.

Results on optic neuritis.

The probable results on optic neuritis of the operation of trephining have been discussed in a paper by Taylor.[1] He showed, by reference to various cases, that not only may the neuritis be relieved by the removal of an intracranial tumour, but that the mere act of trephining in cases where the tumour is out of reach may, by relieving pressure, stop the neuritis. But this is, unfortunately, not always the case, as I have myself had occasion to observe in several instances of this kind.

[1] J. Taylor, "Optic Neuritis in its Relation to Intracranial Tumour and Trephining," *Trans. of the Ophthalmological Society*, vol. iv. (1894) p. 105.

If such palliative trephining is to be performed for the relief of pressure, the opening made in the skull should be a large one, and the dura mater should be fully divided. So far as I am able to judge, the best method of trephining in cases of cerebral tumour is that modification of Wagner's method, devised by J. M. Cotterill and described by him in the *Encyclopædia Medica*, vol. ii. (1899).

Position of trephine opening.

There remains to be considered the question of where the window in the skull should be made, in these cases of palliative trephining. In cases in which, though no approach to exact localisation is possible, there is some suspicion that the tumour may lie at some particular point, it is clear that the opening ought to be made there, on the chance of the tumour revealing itself and proving to be capable of complete or partial removal. But in those cases in which there is no sort of indication of the site of the tumour, or in which the grow this known to be inaccessible, the surgeon should be asked to make his opening over one of the so-called "silent areas" of the brain, so that, in the event of a hernia cerebri resulting, a very important part of the cortex may not be thrown out of function.

When the pressure and distortion of parts produced by the growth of the tumour has interrupted the flow of cerebro-spinal fluid from the ventricular system to the sub-arachnoid space, it may be necessary, in addition to the operation of trephining, to open the lateral ventricle and so allow the fluid to escape.

Lumbar puncture. Sometimes Quincke's lumbar puncture has been tried with the view of relieving intracranial tension in cases of brain tumour, and while in some cases relief follows that procedure, in others it is not successful. This failure is probably due to obstruction of free drainage from the ventricular system to the sub-arachnoid space and spinal meninges. Indeed Oppenheim records a case in which after each tapping the patient experienced an increase of the pain in the head, and one or two instances of sudden death after the operation have appeared in the literature. But, if the operation be performed with due care, such danger is very remote. I have certainly seen the severe pain of these cases markedly relieved by lumbar puncture.

Brain abscess. In regard to abscess occurring within the cranial cavity, it is only necessary to say that

in all cases (save in the metastatic multiple form) when a diagnosis, and if possible a localising diagnosis, has been made, the treatment is purely operative, and should pass into the hands of the surgeon with an indication as to where the trephine opening should be made.

So far, our attention has been directed to the results on the neurone of increase of pressure, sudden or gradual, within the cranial cavity, and the means at our disposal for removing these. When we turn, as we now must do, to the corresponding lesions in the spinal canal, we shall find that the principles which underlie treatment are essentially similar.

Hæmatomyelia. Hæmorrhage into the spinal cord (the condition known as hæmatomyelia) gives rise to a sudden increase of pressure within the spinal canal, and its effects on the upper motor neurone are due chiefly to compression. Its fibres are not, in most cases, torn up by the hæmorrhage, for the extravasation occurs, as a general rule, in the grey matter of the cord. The treatment of this condition is very much that of cerebral hæmorrhage. *Treatment.* Rest is as essential in the one case as in the other, but

in bleeding into the cord conditions of gravitation make us wish to keep the patient lying in the prone posture or on one side, rather than on the back. The hæmorrhage may be arrested, or at least its arrest may be accelerated by venesection in suitable cases,[1] by the application of leeches over the spine, or an ice-bag there, and perhaps by the injection of ergotin. The excitement of the circulation may usefully be stilled by administering morphia hypodermically. Meningeal hæmorrhage should be treated on the same lines. But in this case the use of morphia will be more urgently required on account of the violent pain which usually accompanies such bleeding, and further, if there be signs in a case of meningeal hæmorrhage indicating that the cord is being compressed, it is advisable to call upon a surgeon to perform laminectomy.

Tumours of the spinal cord.

If we except tuberculous disease of the vertebræ and the neighbouring meninges, about which I shall have something to say presently, it may safely be stated that tumours involving the spinal cord are rare. When they occur they produce the usual pressure effects, œdema and

[1] See on page 161 the indications mentioned in connection with cerebral hæmorrhage.

anæmia of the parts affected, and sometimes some inflammation. Among these parts the axones of the upper motor neurone in their way down the pyramidal tracts are notably affected. If a diagnosis of spinal tumour has been reached, the first thing to be considered is whether there is evidence of syphilis or not. In the former case anti-syphilitic treatment should be commenced at once. But even when there is strong reason to doubt the presence of syphilis in the case, it is well to give that treatment a fair trial. A few weeks will generally suffice to show whether improvement is going to take place or not. I have already said, in speaking of intracranial gummata, that treatment with iodide of potassium and mercury is not always completely successful; indeed in some cases the effect is only a very partial one. The same may be said of gummata within the spinal canal. So that in this case too you may fail in curing your patient. If the tumour is not syphilitic, or if, being syphilitic, treatment has not been successful, any other medicinal treatment can only be palliative.

Antisyphilitic treatment.

Therefore, in such a case as this, you have to consider the prospects of an operation for the removal of the growth. But the cases are com-

Operative interference.

paratively few in which an operation has been performed for the removal of a tumour from the

Statistics.

spinal canal. Bruns,[1] in 1897, collected twenty cases from the literature. When Putnam and Warren[2] wrote, towards the end of 1899, the list had risen to thirty-three. Schlesinger[3] adds some twenty-eight cases to this list. The statistics of operation on tumours of the spinal cord performed between the years 1896 and 1902 have been collected by Joseph Collins.[4] He gives the history of seventy cases of the disease, in thirty of which an operation was performed. Of these thirty operations, ten failed, twelve were completely successful, and in eight there was partial success. Tumours of the spinal cord appear to be much more often suited for operation than are those of the brain. The operation will probably become a more frequent one as surgical skill and technique advance. The results, though not very favourable, are better than one might *a priori* expect. Speaking

[1] L. Bruns, *Die Geschwülste des Nervensystems.* Berlin, 1897.

[2] Putnam and Warren, "The surgical treatment of tumours within the spinal canal," *American Journal of the Medical Sciences*, Oct. 1899.

[3] Schlesinger, *Beiträge zur Klinik der Rückenmarks und Wirbeltumoren.* Jena, 1898.

[4] J. Collins, "Spinal-cord tumours," *New York Medical Record*, Dec. 6, 1902, p. 882.

generally, it may be said that about one-half of the cases operated on are either cured or considerably relieved, and that in about one-third of the cases death has resulted from the operation or has been accelerated thereby.

But, in spite of this heavy mortality, it is evident that when antisyphilitic treatment has failed and when a localising diagnosis is possible and has been made, the operation of laminectomy *Laminectomy.* for the removal of the tumour is to be recommended in this otherwise hopeless disease. The risks of the operation should be carefully and conscientiously laid before the patient, for you must remember that fixing the site of a tumour exactly is often very difficult, that a tumour lying on the ventral surface of the cord cannot be removed, and that its situation on the lateral aspect of the cord makes the operation an exceedingly difficult one. When the tumour lies in the substance of the cord, its removal is impossible—that is, impossible without inflicting dangerous damage on the spinal tissue. And the diagnosis as between extra-medullary and intra-medullary tumour is always very difficult to arrive at, and sometimes cannot be reached at all. Such symptoms as dissociated paralysis

of sensation, persistent and bilateral (usually the sense of temperature and that of pain being lost), along with paralysis of the muscles of the upper extremities of the lower neurone type, accompanied by spastic paralysis of the legs, pointing as they do to the presence of a tumour in the centre of the cord, would contra-indicate surgical interference. But even if the tumour is so situated as to render its removal impossible, the operation of laminectomy will relieve the tension within the spinal canal. This appears to be sometimes very beneficial in the case of tumours which cannot be extirpated.

Pott's disease of the spine.

The last of the causes of compression of the spinal cord which I need to mention is tuberculous caries of the vertebral column. Pott's disease is unfortunately a common one, and its treatment requires care, attention, and the exercise of great patience both on your part and on that of the patient. The tuberculous focus is usually in the body of the vertebra, and the caseous matter comes to press on the spinal membranes, and so to compress the cord.

Mechanical effects.

But the first results are due to interference with the vessels and lymphatics of the dura mater—an

interference which leads to œdema of the tissues of the cord. After this has lasted for some time, and in many cases for a very long time, the tissues of the cord soften and break down. It need hardly be said that if treatment is to be effective in restoring the functions of the cord, it must act when only œdema is present and before this softening process occurs. The spinal symptoms of such cases, especially in the earlier stages, are largely those brought on by pressure on the upper motor neurone, namely, spastic paralysis of the limbs.

Treatment.

The treatment, so far as medicines are concerned, is that of tuberculous processes elsewhere. The administration of cod-liver oil should be persistent, and some preparation of iron should also be given—preferably, I think, the syrup of the iodide of iron. The food should be plentiful, nutritious and plainly cooked. So far as may be consistent with the other parts of the treatment, the patient should lie in the open air as much as possible.

Apart from these points the main treatment is one of rest, the object being to prevent movement in the spine and to take off pressure from the focus of the tuberculous process.

These ends are best attained by confining the patient to bed for many months, securing immobility of the spine and relieving pressure by means of extension.

Extension. The easiest and, in my opinion, the best way to bring about the extension necessary in such cases is to place a leather band, properly padded, round the occiput, the lower jaw and the chin, to connect it behind each ear with straps which, passing up either side of the vertex, are attached to a square piece of wood sufficiently wide to keep the pressure of the straps off the sides of the head. To this square of wood is attached a cord which, passing over a pulley fastened to the head of the bed, supports at its other end the *Details of method.* weight required for extension. If the head of the bed be raised on wooden blocks, the lower part of the patient's body, tending to slip down, is sufficient to counterbalance the weight used in extending the head and upper part of the spine. If the caries is in the dorsal or lumbar regions of the spine, extension should also be applied by means of straps passing under the axillæ. In the former case a weight of only a few pounds can be borne, in the latter the weight may be gradually raised to twenty pounds or more.

Such extension, to be effective, has to be maintained for many months, and during this long period the condition of the bladder must be sedulously watched, and the greatest care is necessary, in nursing, to avoid the occurrenee of bed-sores. I have found it well to use, in sueh cases, a somewhat thin mattress, and to make its surfaee more truly a plane by laying beneath it a broad wooden board. This avoids the formation in the bed of a deep depression under the hips of the patient. Such a depression obviously interferes with proper extension in that it does not allow the weight of the lower body to tell fully and properly. After the extension has done its work, and the patient is recovering, it is well to apply a poro-plastic jaeket or other similar support. This will keep up the immobility of the spine to a certain extent, and at the same time allow the patient to move about in the open air.

Attention to bladder.

Treatment by extension is usually successful with children and in young adults, and I have seen wonderfully good results even in apparently unfavourable cases. I recall the case of a patient of this kind twenty-four years of age, who had caries in the lower dorsal region with well-marked

Favourable results of extension.

pressure symptoms, some anæsthesia, spastic paraplegia, and greatly exaggerated deep reflexes. After about a year of constant extension she was so well that I sent her to the country and allowed her to go about in an ordinary way. Some months later she returned to report her progress. I found then that she had construed her orders somewhat more liberally than one would have thought of, and had been working off her animal spirits at many dances. This exercise, for which she had an inordinate affection, must have been beneficial, for she was then, and is now, in excellent health.

Of late years the old practice of sudden and forcible reduction of the spinal curvature has been revived by Calot. This method is not correct in theory and works badly in practice.

Laminectomy.

If extension fails, then our only resource is in the operation of laminectomy, combined with scraping out, disinfecting and draining the tuberculous cavity. This operation, the introduction of which we owe largely to Victor Horsley, to whom great credit is due in this regard, is not as a rule a dangerous one. But the after-treatment is exceedingly tedious and troublesome, for if the body of a vertebra be diseased, then in

removing the laminæ the surgeon takes away the remaining support of the vertebral arch, and may be conceived of as separating the spinal column into two parts. Nevertheless the operation is one which is sometimes necessary, and that chiefly under three conditions: first, in cases in which the disease is high up in the spine and threatens to interfere with respiration; second, when there are signs of abscess formation and consequent increase of pressure and risk of infection of the cord; and third, when extension has failed to improve matters, and when at the same time there is no reason to believe that myelitis is present.

Indications for operation.

The site of the lesion and the condition of respiration afford sufficient guidance under the first of these headings. The presence of an abscess is difficult to ascertain when the disease affects the body of the vertebra, though easy when the lamina is its seat. Sudden increase in the paralytic symptoms along with a pyrexia which is not traceable to tuberculous disease elsewhere, will suggest the formation of an abscess sufficiently to justify the performance of an operation.

Except in very special cases a full trial ought

to be given to the treatment by extension before any operative measures are considered. If this treatment is going to succeed, the patient will show improvement in two or three months. And in most cases such waiting does no appreciable harm, for the œdema of the cord produced by pressure may cause paraplegia for many months without doing such damage to the cord as cannot be recovered from.

Laminectomy should not be attempted in those cases in which there is evidence of active disease elsewhere. It need hardly be added that when the seat of the disease is in the lamina, the operation is fully justified and very advisable.

We have now considered the treatment of such diseases of the upper motor neurone as are due to toxines, and that of those which are due in some considerable measure at least to increase of pressure within the cavity of the cranium and the spinal canal. There remains for discussion one other prominent cause of such disease, namely, destruction from softening, the result of occlusion *Cerebral softening.* of a cerebral vessel. This may occur from thrombosis or from embolism. An apoplectic attack arising from one or other of these two

causes is not to be treated in the same way as one due to cerebral hæmorrhage. The diagnosis must therefore be carefully established before treatment can be safely or usefully directed.

Treatment. When a cerebral vessel has become occluded neither venesection nor strong purgation should be thought of. The patient is to be kept at rest, the bladder attended to and the occurrence of bed-sores guarded against, in the manner previously described. Otherwise, the treatment depends on the state of the circulation. Alcohol is frequently needed, and it may be necessary to give digitalis or strophanthus. Strychnine may also be required, and sometimes nitro-glycerine or erythrol tetranitrate.

During recovery, massage and passive movements of the paralysed limbs will prevent fixation, and the application of a light splint to the hand and arm at night may keep the contracture from becoming severe. As time goes on the patient should be encouraged to make active movements. If there be much restlessness, the bromides should be given, and even opium may be required. The administration of iodide of potassium is usually of service in the later stages.

In cases of thrombosis in which a syphilitic

endarteritis is the cause, vigorous antisyphilitic treatment has often a very striking curative effect.

General points in management of upper motor neurone paralysis.

In conclusion, a few words may be said as to the management of certain of the special symptoms which result from that descending degeneration of the upper motor neurone which is produced by the various lesions we have been considering.

The muscles involved are paralysed, and this paralysis is more or less complete according to the variety of the lesion in question, according to its severity and completeness, and according to the part of the body which is affected. In cases in which the paralysis is not complete, when the disease has reached the chronic stage, a certain amount of mild gymnastic exercises does good, provided no undue fatigue results. The use of the "go-cart," for example, is very useful in some comparatively mild cases of myelitis, and in the same class of cases gentle massage will be of benefit. Certainly graduated and careful exercises such as I shall have to speak of in connection with the treatment of tabes, may do some good in these cases. It has, however,

Gymnastics.

Graduated exercises.

been already pointed out that the vicarious action of other neurones, possible in cases of lesion of the centripetal co-ordinating apparatus, is hardly possible in those of the pyramidal tracts which we have been considering. In these there are no alternative paths, no neurones capable of acting vicariously; for the vestibulo-spinal and the rubro-spinal paths can hardly take over the work of the great cortico-spinal tracts.

Where a certain amount of atrophy, from disuse or from involvement of the lower motor neurone, has occurred in muscles which still remain somewhat under the power of the Will, faradism may be cautiously tried, say once daily for five minutes. This treatment is often a most useful one, but great care should be taken not to apply the current to such muscles as are in a spastic condition. An application of this kind would excite and increase their rigidity, and in a case of myelitis it might influence unfavourably the focus of disease in the cord.

But paralysis is not the only result of degeneration of the upper motor neurone. We have also to combat the contracture of *Contracture.* the muscles, and the tendency to spasmodic

reflex jerking whieh sometimes show themselves. These are very difficult to treat with sueeess. In hemiplegia the tendeney to contracture may be met to some extent at least by bathing the limb with hot water, and by gentle frietion of the skin from below upwards. No strong massage of the paralysed musèles should be permitted, for this tends to inerease the eontracture. Passive movements should be made at least twiee a day, and some good usually results from stimulation of the antagonistie museles with the faradie eurrent. It is also advisable to apply a light splint to the arm and hand at night, as I have already told you.

In eases presenting the symptoms of lateral selerosis the spasm and stiffness of the muscles are very difficult to deal with. This stiffness relaxes at onee in a warm bath, but it returns in a very short time after the bath is over. Gentle frietion and slow passive movements may be expeeted to produee good results, and in some cases gentle gymnastic exereise tends to give greater freedom to the museular movements.

The most distressing of all this elass of symptoms are the spontaneous, or apparently

spontaneous jerkings of the limbs which occur in chronic myelitis. Some patients suffer much by reason of these, and unfortunately you will find that they are very difficult to control. As a rule drugs do not act well, but you may try full doses of bromide of potassium given along with the iodine salt. Atropine or hyoscine may also be tried. Warmth certainly tends to diminish these spasms, and you may sometimes observe how the spasmodic jerking increases when you remove the bed-clothes to examine the legs, and so allow the cold to act. A warm bath gives great relief, and when the condition is causing much distress it may be well to place the patient for some hours, or even for some days, in a permanent warm bath. In some cases, however, the distress caused by these jerkings is so great that morphia may have to be used. *Muscular jerkings.*

Tremor is another symptom liable to occur in such cases, and fitted to cause great discomfort to the patient. Frequently seen in alcoholism and in cases of metallic poisoning, it is there to be treated by the removal of the cause, after which the tremor usually ceases. The intention-tremor of disseminated sclerosis may sometimes *Tremor.*

be controlled by giving small doses of strychnine. Hyoscine would also in some cases diminish the tremor, but the advisability of giving that remedy is doubtful. When we come to talk of the treatment of locomotor ataxia I shall have a good deal to say as to the methodical re-education of such patients in the art of making co-ordinated movements. Treatment of a similar kind is to a certain extent available in disseminated sclerosis, though, for reasons I have already given you, such treatment is not at all so successful in cases of lesion of the pyramidal tracts as in those of the centripetal co-ordinating apparatus. You will remember that the tremor and oscillation in this disease increase in amplitude as the voluntary movement is being made. The patient should be instructed to practise such movements as he must frequently have to make in daily life, and to practise each movement many times in succession, performing it slowly and with every attempt at precision. In this way he may recover, to some extent at least, the power of making ordinary movements with steadiness.

We have now completed the consideration of the more important of those maladies which pro-

duce paralysis of the upper motor neurone. These have been taken as types of the various lesions affecting the pyramidal tracts, and from a consideration of the lines of treatment to be adopted in them, it will not be difficult to see what the treatment should be in those less common disorders which have not been specially mentioned.

In the next lecture we shall consider the treatment of those diseases which are characterised by the phenomena of irritation of the upper motor neurone.

LECTURE VI

ON THE TREAMENT OF IRRITATIVE DISEASES OF THE UPPER MOTOR NEURONE

Signs of irritation of the upper motor neurone—Their causation—Idiopathic epilepsy—Its various causes—Its prophylaxis—Its treatment—General measures—Treatment by bromides—Metatrophic method—Operative treatment—Symptomatic epilepsy—Its treatment—Infantile convulsions—Tetany and its treatment—Chorea, its causes and treatment.

Symptoms. THE symptoms due to irritation of the upper motor neurone are usually distinet, and even striking. Convulsions, eomplete—or partial—athetosis, ehorea, are eonditions whieh are readily reeognisable by the physieian, and whieh strongly impress and engage the mind of the patient and his friends.

But while, from a elinieal point of view, these are salient features in any case in whieh they oeeur, from the side of pathology they frequently present great obscurity. In this respeet they eontrast in a marked way with

Obscurity of their causation.

those lesions of this neurone which produce paralysis. In the latter, the method of origin of the lesion and the way in which it acts in causing the paralysis are in most instances fairly clear to us. In cases of irritation of the neurone there is seldom such clearness.

Idiopathic epilepsy.

Let us, in the first place, consider cases of so-called idiopathic epilepsy, a group which is gradually being diminished in numbers as the causation of the disease is becoming better understood. In spite of the observations of Kussmaul and Tenner, of Nothnagel, of Luciani, of Unverricht, as well as the more recent experiments of Prus,[1] which point to the origin of the disease in the central ganglia, the pons and the medulla, the view is now generally held that the convulsions which characterise epilepsy are the outward expression of explosions of nerve energy in the grey matter of the cerebral cortex. The acute clinical observations of Hughlings Jackson in elucidating the pathology of partial con-

Theory of causation.

[1] Prus, "Ueber die Leitungsbahnen und Pathogenese der Rindenepilepsie," *Wiener klin. Woch.* 1898, p. 856; also, "Ueber elektrische Reizung der Vierhügel," in the same journal, 1899, p. 1124. Prus also holds that the impulse which causes muscular spasm when the cortex is stimulated does not pass down the pyramidal tracts, but traverses certain other fibres, which he calls the "extra-pyramidal tract."

vulsions, taken along with the experiments of Fritsch and Hitzig, who showed that such convulsions could be artificially produced by stimulation of the cortex, leave little doubt in the mind as to the mechanism of the general disease. But while this is so, it is not easy to understand the method of production of these explosions in cases of purely idiopathic epilepsy.

Inherited predisposition.

Of one thing there can be no doubt, namely, that there must be a certain predisposition. The hereditary character of the disease is, indeed, usually well marked. There is a lessened cortical stability in these cases; and in the family history of an epileptic there will usually be found individuals who have suffered from that disease, from insanity, from alcoholism, or from some other of those disorders which specially characterise neurotic families. I cannot tell you with certainty whether consanguinity on the part of the parents is of itself a cause of such cortical weakness, but it is certain that if the family from which both parents spring has had epileptics among its numbers, such a consanguineous marriage would be exceedingly likely to be followed by epilepsy in the children.

Susceptibility of upper motor neurone.

As I have already pointed out,[1] the upper motor neurone is late in development, is highly specialised, is delicate, and is very susceptible of injury. It is probably on that account that this neurone so often shows traces of the severe strain which falls on the tissues generally at the time of puberty, especially in the case of the female. A large proportion of the instances of epilepsy we meet with arises at this period of life—a fact which has an important bearing, as we shall see presently, on the prophylaxis of the disease.

Proximate causes of epilepsy.

But predisposition and the lessening of cortical stability is not of itself enough to account for the onset of epilepsy. There must also be an exciting or proximate cause. A sudden fright or other strong mental emotion or impression, may start off a series of explosive cortical discharges, and lead to a life-long epilepsy.

Peripheral irritation.

Peripheral irritation may also occasionally, but I think rarely, act as the exciting cause of the disease in those predisposed. The presence of tape-worm in the intestine, for example, may so act, and the reflex irritation of a scar on some part—it may be a distant part—of the body

[1] See the preceding lecture.

may exceptionally act as the starting point of the disease. Many years ago Brown-Séquard showed that injuries of various parts of the brain and cord, and even of the sciatic nerve might, in guinea-pigs, produce epilepsy. He was under the impression that the disease, so created, transmitted itself to the offspring, but this does not appear to be the case.

Brown-Séquard fits.

In a certain proportion of cases epileptic seizures are the direct sequence of convulsions from which the patient suffered in infancy, and in these instances the effect of rickets as a predisposing cause is very strong. It seems indeed as if the cortex, having once become accustomed to discharge in the violent fashion of epilepsy, did not readily lose that habit. There are many cases on record in which, though the epilepsy undoubtedly proceeded from some evident source of irritation, the fits did not cease at once on the removal of that irritation, but persisted until the cortex had been quieted down by medical treatment.

Toxic causes.

Another potent cause of the production of epilepsy, and one which will, I believe, be more recognised and understood in the future, is the action of poisons on the cortex. Alcohol is

typically one of these, and its misuse, if persisted in for long, is apt to be followed by epileptic attacks. In a few of these cases of alcoholic epilepsy,[1] occurring after middle age, the poison probably acts by the medium of the arterio-sclerotic changes which it produces in the vessels of the brain, leading in this way to circulatory disturbance and deficient nutrition. Perhaps the cardiac changes which follow the abuse of alcohol may also assist in its production.[2] In this form of alcoholic epilepsy the fits do not disappear when the patient ceases to take the alcohol. Much more commonly, however, the alcohol acts purely as a toxine, causing thereby the cortical explosions of epilepsy; and this action is usually seen about the same period in the case as that at which symptoms of neuritis show themselves, or the signs of delirium tremens appear. This, the commonest form of alcoholic epilepsy, is usually readily cured by removal of the poison, the fits generally ceasing within a fortnight of the commencement of total abstinence.

Alcohol.

[1] See a paper by Bratz, "Alkohol und Epilepsie," *Allgem. Zeit. f. Psychiatrie*, 1899, p. 334.

[2] A. Smith, "Ueber eine . . . als 'alkohologene cardiale Epilepsie' charakterisirende Gruppe epileptoïder Zustände," *Mün. med. Woch.* Bd. 45 (1898), p. 1372.

Absinthe. Absinthe is a cause of epilepsy even more potent than alcohol; but fortunately this dangerous nerve-poison has not yet invaded our country to any extent. Chronic lead-poisoning is another of those conditions by which the tendency to epilepsy in a predisposed brain may be excited.

Syphilis. Certain of the toxines of infective disease may also act in this way. Syphilis certainly does so sometimes, though in many syphilitic epileptics the fits are probably due to those arterial changes to which that disease so often gives rise. *Typhoid fever.* The toxine of the typhoid bacillus, as Friedländer has shown,[1] is a potent, though not a very frequent, cause of epilepsy, and probably many other like toxines act in the same way. Eclampsia in childhood often arises in this fashion, and it seems probable that the fits of infancy not uncommonly translate themselves into the epilepsy of the adolescent and the adult.

Auto-intoxication. But of all the causes of that form of epilepsy which we call idiopathic, probably the most common is auto-intoxication, in which the

[1] Friedländer, "Ueber den Einfluss des Typhus abdominalis auf das Central-nervensystem," *Monatsschr. f. Psych. und Neur.* 1900, p. 333.

toxines which act on the predisposed cortex are certain bodies formed in the gastro-intestinal canal by reason of digestive disorders, or elsewhere in the body by faulty metabolism. What these substances are we do not know exactly, though something is known as to their solubility in various media and experimentally as to their toxic power. The whole subject is a very obscure one, and I cannot discuss it fully here. Much work in this line has been done of late years, particularly in France and Italy, towards its elucidation. It is not possible at present to arrive at any very definite conclusion, beyond the general one that such poisons are formed within the gastro-intestinal canal, that they are absorbed into the blood, and that they thus reach the cortex. Before the fit occurs the nitrogenous elimination is decreased,[1] as well as that of the chlorides and P_2O_5, while after the fit is over all the waste substances of metabolism are increased. When the toxicity of the urine and of the blood is tested by injecting them into animals, their poisonous quality is said to be considerably increased after a fit. There is also

[1] Froehner and Hoppe, "Der Stoffwechsel von Epileptikern unter dem Einfluss der Schilddrüsenfütterung," *Psych. Woch.* 1899, S. 313.

evidence, in the increased excretion of etherial sulphates, that just before a fit comes on, in many cases of epilepsy, there is an increased amount of putrefactive change in the stomach and intestines.

Bra,[1] investigating the blood of seventy patients suffering from epilepsy, discovered a micrococcus which, present in some numbers just before and during the fit, was rarely to be found during the intervals. This "neurococcus" he believes to be the cause of epilepsy, regarding that disease as analogous to malaria, the convulsions corresponding to the attacks of fever. He found that cultures of this micro-organism gave rise to convulsions, when injected intravenously in rabbits.

Cholin. It may be that cholin plays a part in the causation of epilepsy. Donath[2] found that substance was present in some quantity in the cerebro-spinal fluid of almost all the cases he investigated. Cholin is capable of producing convulsions when brought in contact with the brain substance of normal dogs. Perhaps, there-

[1] Bra, "Du parasite trouvé dans le sang des épileptiques," *Revue Neurologique*, t. x. (1902), p. 447.

[2] Donath, "Das Vorkommen und die Bedeutung des Cholins in der Cerebrospinalflüssigkeit bei Epilepsie," *Zeit. f. Phys. Chemie*, Bd. 39 (1903), H. vi. S. 526.

fore, we may look upon this as, in some cases at any rate, the proximate cause of the cortical explosion. Mansfield,[1] however, has criticised Donath's test in an unfavourable sense.

Prophylaxis

Before considering the treatment of idiopathic epilepsy, a word may be said as to prophylaxis as applied to the children of an epileptic parent, such of them at least as show signs of a neurotic inheritance. In the first of these lectures I have indicated the general lines on which such a child should be brought up, and little remains to be added. If a child of epileptic parentage has inherited a tendency to that disease, he will probably show signs of such in the shape of convulsions when any considerable temporary irritation occurs—in teething, for example, or during some febrile complaint. In these circumstances very special attention should be paid to the rearing of the child. He should be much in the open air, have plenty of exercise, little brain work, plenty of sleep. His food should be ample, but plain and light, and it should include but little butcher meat. When the

[1] Mansfield, "Ueber den Donathschen Nachweis von Cholin," in the same journal, Bd. 42 (1904), S. 157.

time of puberty arrives, special care should be taken to avoid brain fatigue and emotional excitement.

Treatment of epilepsy.

The treatment of a patient suffering from idiopathic epilepsy is one which demands great care and patience. In the case of adults it is well, when the diagnosis has been made, to inform the patient frankly that even under the most favourable circumstances he must remain under medical observation and control for some years, that he will have to exercise considerable self-denial in following rigorously the rules laid down for him, but that the disease need not materially affect his work, nor need it take from him many of the pleasures of life.

General considerations.

Diet.

The diet of an epileptic should be light and easily digestible. Its composition must, of course, vary somewhat according to the individuality of each patient. But on the whole it should consist largely of milk, farinaceous food and vegetables, and should include but little meat. The great object is to prevent digestive disturbance and auto-intoxication, which, as has been already said, may give rise to explosions of cortical energy. The chief meal should be taken

in the middle of the day and the supper should be a light one. As a rule, no alcohol,[1] tea, coffee or tobacco should be made use of. The condition of the bowels must, of course, be attended to and no constipation allowed to occur. The patient should take as much exercise in the open air as may be possible without inducing undue fatigue.

Mental work. The mental work of an epileptic patient should be light, but in many cases the education of a young patient need not be materially interfered with, though it must of necessity be conducted at home. *Occupation.* The occupation of an epileptic should clearly be one which does not expose him to special risk of traumatism during his fits.

Rest and massage. When a severe case of epilepsy first comes under observation with a view to treatment, you may find it to be a good plan to confine the patient to bed for a month or more, supplying the need of exercise by means of a moderate massage. In this way you can keep him under more careful observation than might otherwise be possible, and undoubtedly the rest and quiet-

[1] German writers strive hard to show that beer is rather helpful than harmful in these cases, and Bratz has recently stated (*Monatsschr. f. Psych.* 1901) that in the case of two hundred epileptics under his observation, two bottles of beer in the day had the effect rather of decreasing the number of fits than of increasing them. It is probably best, however, for an epileptic to be a total abstainer.

ness of life in bed allows your treatment every chance of success.

Thyroid feeding. In cases in which there is evidence, from examination of the urine, that the metabolism of the body is upset, and that nitrogenous elimination is being curtailed, it may be well to try the effects of thyroid feeding. In this way the nitrogenous excretion may be largely increased, and some cases are much improved by this form of treatment.

If you have reason to believe that putrefaction of food in the gastro-intestinal tract and consequent toxine poisoning, is the starting point of each fit (and the quantities of etherial sulphates in the urine will guide you in this matter), then clearly you will try by purgatives and enemata, and by washing out the stomach, to get rid of these products. The administration of salol and of small doses of mercury may also help.

Purgatives.

Lavage of stomach.

Counteracting and removing poisons. Cases which arise from the abuse of alcohol, from chronic lead-poisoning and from syphilitic infection must be treated in the manner already described as appropriate to these conditions.

Sources of reflex irritation. It need hardly be added that in cases in which the attacks proceed from the reflex irritation of intestinal parasites or other removable cause, due

means should be taken to free the patient from such sources of irritation.

Treatment by bromide salts.

But by far the most important line of treatment in idiopathic epilepsy is that by means of the bromides. It seems probable that these act on the cerebral centres by replacing the chloride of sodium in which these tissues are normally bathed. Beyond this we know nothing as to the *modus operandi* of their action. That the action is a most powerful and remarkable one, you must all have had opportunity of convincing yourselves.

Prejudice against their employment.

There exists, unfortunately, in the public mind a strong prejudice against the use of bromide, which, if prolonged, is held to produce certain marked signs of mental deterioration. This is a distorted and incorrect view. It is not, of course, natural that the chloride salt, which is normally present in the cells of the cortex, should be displaced by the bromide; and I suppose that a long continuence of this condition might be expected to produce some effect on the function of the cortex. This we may frankly admit. But I am very sure that those signs of mental deterioration, which so strike the observer, are the outcome, not of the administration of

bromide, but of the epilepsy for the cure of which that bromide was given. They point rather to a deficiency in the quantity administered than to any untoward action of the drug.[1]

The bromides of potassium, sodium and ammonium appear to be about equal in therapeutical value. If one were to indicate any preference, it would perhaps be for the bromide of potassium. But some people prefer to give a mixture of the three salts. Of late the bromide of strontium has had some vogue, but it does not appear to possess any distinct advantage,[2] and it is considerably more expensive than the others. The use of camphor monobromate has been strongly recommended, particularly by French physicians.[3] It is said to be specially suited for cases of *petit mal*. I cannot speak, however, from any extended experience of the use of this drug.

Selection of bromide salt.

It is usual in the case of adults to begin by administering forty-five grains in each twenty-four hours, and it is well to give this quantity

Dose to be given.

[1] On this point see the remarks of Risien Russell, *Transactions of the Medical Society of London*, 1903, p. 125.

[2] J. G. Smith, "Note on twelve cases of epileptic insanity treated by means of bromide of strontium," *Lancet*, 1899, Aug. 12th.

[3] Bourneville et Ambard, "Nouvelle contribution à l'étude de l'épilepsie vertigineuse et à son traitement par le bromure de camphre," *Archiv. de Neurologie*, t. xiv. (1902), p. 1.

in divided doses, three times a day. If this is not sufficient to stop the fits, the quantity administered should be increased gradually until the necessary dosage is ascertained. This is always a matter of experience in each individual case. But it is well to remember that if you do not attain the desired result with a daily quantity of 110 grains in the case of an adult, or 50 or 60 grains in a child of ten, little good is to be expected from raising the dose above these limits. While as a general rule I prefer to give the bromide salt in divided doses, yet in some cases it is better to administer the total quantity for the 24 hours in one dose. This method is indicated in cases in which the fits come at some particular hour. The bromide should then be given about two hours before that time.

Results of bromide treatment.

In most cases the epileptic attacks are more or less checked and in many they entirely disappear during bromide administration. In a small percentage a complete and permanent cure is effected. The bromide has, however, to be given for a long period, and the administration should be absolutely continuous, no intermissions being allowed to occur save in exceptional cases.

And if a cure has, happily, been effected, the continuous use of the bromide should go on for at least two years after the last fit. It may then be gradually reduced and its use ultimately discontinued.

Bromism. The disagreeable signs of bromism do not, as a rule, occur if the daily dose does not exceed 60 grains in adult cases, or 40 grains in the case of children. With quantities above this, however, such symptoms frequently arise. There is then a certain amount of mental torpor and bodily weakness, and the superficial reflexes become diminished and ultimately lost. It used to be held, indeed, that the disappearance of the palate and pharyngeal reflex was a sign of full bromide action. But it is now known that this reflex is not always present in normal people. *Action on reflexes.* Ziehen has therefore suggested that the corneal reflex should be employed as a measure of the saturation of the body with bromine. If that reflex fails in one or in both eyes, the bromide treatment should be stopped for the time, or its poisonous effect may be inhibited by the administration of chloride of sodium.

Disturbance of digestion. The digestion is apt to be disturbed and may be so up to the point of absolute loss of appetite

and loathing of food. This is usually accompanied by marked fœtor of breath resulting from decomposition of the buccal contents. Such disturbance is best met by regulating the diet, by giving purgatives, and by administering such intestinal antiseptics as salol, salicylate of bismuth, or small doses of mercury.

Acne.

Another most disagreeable symptom of bromism is the eruption of acne which is so often seen, and which may in bad cases proceed to extensive infiltration and necrosis of the skin. This complication is best met by the administration of small doses of arsenic along with the bromide salt.

Conditions of success in bromide treatment.

It has been said that some cases of idiopathic epilepsy do not improve under the influence of bromide treatment or do so only very temporarily. Have we any guide to help us in deciding which cases are likely to improve under such treatment and which are not?[1] There is, first of all, a class of case which occurs in comparatively young persons, in which the fits, while not distinguishable from those of true epilepsy, are accompanied by various paræsthesiæ and

[1] A most thoughtful paper on this point has recently been published by Fürstner, "Ueber Behandlung der Epilepsie," *Archiv f. Psych. und Nervenkr.*, Bd. 33, S. 240.

other sensory disturbances. These fits are apt to be associated with menstruation or other occasional irritation. Such cases are really hysterical. They derive no benefit from bromides—are indeed sometimes the worse of such treatment—but they at once improve under the moral regimen of an hospital or nursing home.

Late epilepsy. That form of the disease which is known as "late epilepsy,"—those cases, that is, in which the fits appear for the first time late in life,—are not very amenable to bromide treatment. A small percentage do well, and these are probably the more purely functional cases. Bromides have little or no effect in those instances of late epilepsy which depend for their causation on arterio-sclerotic changes in the cerebral vessels.

Metatrophic treatment. An interesting modification of the bromide treatment is that metatrophic method which has been suggested by Richet and Toulouse.[1] By it the objects of bromide administration are attained with much smaller doses than are otherwise necessary. It has already been said that in acting on the grey matter of the central nervous system the bromide salt probably does so by dis-

[1] Richet and Toulouse, "Effets d'une alimentation pauvre en chlorures sur le traitement de l'épilepsie par le bromure de sodium," *Comptes rendus de l'Académie des Sciences*, Nov. 20, 1899, p. 850.

placing the chloride, and I have also alluded to the power of chloride of sodium in inhibiting the action of the bromide salt, and the usefulness of its administration if that action appears to be going too far. It is therefore not unlikely that the action of the bromide salt will be the more powerful the less chlorides are contained in the tissues. The theory on which this method rests was, as Risien Russell has very properly pointed out, originally formulated by Hughlings Jackson. To Richet and Toulouse is due the credit of its practical application. The point which they specially investigated is whether, by largely diminishing the quantity of chlorides taken with food, a smaller dose of bromide would be adequate to arrest the fits. They proceeded to test the practical value of this method in the case of thirty epileptics who were placed upon a special diet poor in table salt. It was then found that comparatively small doses of bromide (thirty grains a day) were sufficient to arrest the fits. And the special value of this interesting method of treatment is shown by the fact that when those persons returned to normal diet—the dose of bromide remaining the same—the fits at once recurred. This method, so far as I have been able

Theory on which this rests.

Diet poor in chloride of sodium.

to judge, is so well worthy of trial that I shall now give you the details of the diet Richet and Toulouse recommend. It is as follows:—

Milk . .	1000	grammes.
Meat . .	300	,,
Potato . .	300	,,
Flour . .	200	,,
Two eggs .	70	,,
Sugar . .	50	,,
Coffee . .	100	,,
Butter . .	40	,,

The quantity of chlorides, estimated as chloride of sodium, which this diet contains, is about 2 grammes, contrasting with the 10 to 15 grammes which are found in ordinary diet. This restriction of chlorides does not seem as a general rule to produce any disagreeable effects, and if such were to occur, it would be easy for you to add a little salt, say 3 to 5 grammes, to the diet, a quantity which would not materially interfere with the treatment. But you will readily understand that the bromide action has to be watched with great care in such cases.

Modifications of this method.

This method, or modifications of it, has been tested by many observers and on the whole with marked and encouraging success. My own experience is favourable. Eason, after careful

study,[1] came to a like conclusion. He recommends that the bread given to the patient should be specially made with phosphate of sodium instead of common salt. Balint[2] goes a step further, and gives bread made with bromide of sodium, and which he calls "Bromopan." Meyer[3] and others have followed this plan and report favourably on it. I have not myself tried this bread in any case of epilepsy, and from what I have myself seen I am inclined to think that a sufficient degree of hypochlorisation is to be reached by a diet on the lines originally recommended by Richet and Toulouse.

Flechsig's method.

The method of Flechsig in which he alternates courses of bromide with courses of opium is not one to be recommended.

Other preparations of bromide.

Of late the use of one or two other bromide preparations has been suggested. The best of these is bromipin, which is prepared by adding bromine to sesame oil in the proportion of 10 per cent. One tablespoonful therefore contains 24 grains of bromide, which is chemically equal

[1] Eason, "Observations on a case of epilepsy," *Scottish Medical and Surgical Journal*, vol. xi. p. 123.

[2] Balint, "Weitere Beiträge zur diätetischen Behandlung der Epilepsie," *Neurol. Centralblatt*, 1903, S. 347.

[3] Meyer, "Zur diätetischen Brombehandlung der Epilepsie," *Berliner klin. Wochenschrift*, 1903, S. 1049.

to 38 grains of potassium bromide, but is rather more powerful physiologically and therapeutically. The taste is oily and disagreeable to some patients. While it is said to be less liable to cause bromism than the alkaline salts, its chief advantage seems to be found in the case of those epileptics who refuse to take bromides. The bromipin may then be given in soup or in coffee without the knowledge of the patient.

Bromipin.

Sometimes you will find that it is advisable to add other drugs to the bromide you are giving, so as to enhance its action. The addition of five minims of tincture of digitalis is recommended by Gowers, and this sometimes proves useful, as does also the combination of belladonna with the bromide. Bechterew[1] uses a combination of bromide with infusion of Adonis vernalis.

Addition of digitalis.

And of belladonna.

The substitutes for bromides are of comparatively little service. Nitrate of silver used to be given years ago, before the advantages of the bromide treatment were discovered. It is not now employed. Gowers recommends the use of bi-borate of sodium (5 to 10 grains) in cases

Substitutes for the bromides.

[1] Bechterew, "Ueber die Bedeutung des gleichzeitigen Gebrauchs der Bromide und der Adonis vernalis bei Epilepsie," *Neurol. Centralbl.*, Bd. 13 (1894), S. 838. See also a paper by the same author, "Ueber die Bedeutung der Cardiaca bei der Behandlung der Epilepsie," in the above journal for 1898, p. 290.

unsuitable for bromide treatment, and oxide of zinc, an old anti-epileptic remedy, still proves of some service occasionally.

Treatment of the epileptic convulsion.

I have not hitherto spoken of the treatment of the attack itself. In very rare cases it may be possible by sudden stimulation to abort the convulsion. When, for example, the aura commences in one hand, the patient may prevent the full development of the attack by suddenly tightening a strap round the arm above the point at which the aura starts. Indeed some patients in whom the power of Will is still strong, are able to avert the attack by a powerful mental effort. No doubt this power is capable of cultivation[1] but it can hardly come into useful action unless an aura gives distinct and timely warning of the approaching convulsion. But once the attack really commences, there is little to be done beyond loosening the clothing round the neck of the patient, preventing the tongue from being bitten, and if suffocation threatens, drawing the tongue forward. It is well to see that the post-epileptic sleep is undisturbed.

[1] See paper by H. Campbell Thomson, "The treatment of epilepsy by psychical methods." *Lancet* (1903), vol. i. p. 1092.

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Status epilepticus.

If, however, the patient passes into that series of convulsions which we know as the "status epilepticus," life is in great danger and every possible means should be used to stop the attack. The administration of chloroform or of ether is then indicated. Chloral or amylene hydrate should also be given, by the mouth if possible, or by the bowel; but if the patient, as often happens, cannot swallow and cannot retain enemata, then the amylene hydrate may be injected into the muscles. Nitrite of amyl or nitro-glycerine sometimes does good, and occasionally hypodermic injection of morphia may be required. In the status epilepticus this is a somewhat dangerous proceeding, and therefore the dose of morphia should be small, not more than one-twelfth of a grain. If hyperpyrexia threatens, the use of the cold pack or of a cold bath is indicated.

Use of cocaine.

In a grave case where death is threatening from the long series of convulsions in the status epilepticus, it may fairly be considered whether an intra-dural injection of cocaine might not be tried. According to Jacob,[1] medicines which are

[1] Jacob, "Klinische und experimentelle Erfahrungen über die Duralinfusion," *Deutsche med. Woch.*, 1900, p. 46.

introduced into the spinal dura are diffused by the cerebro-spinal fluid, and he believes that they are in this way conveyed to the brain. The observations of Bier,[1] in his operations on the human subject, fully confirm this ; indeed he has to adopt special measures to prevent the cocaine which he has injected into the spinal meninges from reaching the brain. We know from the experiments of Prus[2] that in animals the painting of a solution of cocaine over the motor cortex prevents the convulsions to which faradic stimulation would otherwise give rise. Therefore in a case of dangerous infantile convulsions it might perhaps be permissible to inject cocaine in a weak solution through the fontanelle. In like manner, in an adult, if the status epilepticus appeared about to end fatally, it might be justifiable to make a small trephine opening and apply the cocaine solution directly to the motor cortex.

Operative treatment of idiopathic epilepsy.

Passing now to the operative treatment of epilepsy, it may be said in the first place that in those rare cases in which a neuroma or an old

[1] Bier, "Weitere Mittheilungen über Rückenmarksanästhesie," *D. Arch. f. kl. Chir.* Bd. 64, H. 1.
[2] Prus, *loc. cit.*

cicatrix is the starting point of the aura or evidently the reflex cause of the attack, the removal of such peripheral irritation is clearly indicated.

Various singular operations.

In cases of pure idiopathic epilepsy surgical treatment is of little avail; indeed it is somewhat amusing, if a little sad, to contemplate the various forms of operation which surgeons have, from time to time, devised and carried out for the cure of epilepsy. They have performed tracheotomy in the hope of effecting a cure; they have ligatured the carotids or the vertebral vessels; they have extirpated the superior cervical ganglion of the sympathetic; they have performed the rite of circumcision; they have castrated people whose testicles were not diseased; they have excised the clitoris, and removed healthy ovaries; indeed they seem to have left nothing undone, save the cure of the disease for which these ingenious and interesting operations were undertaken.[1]

Trephining.

There is something, however, to be said for trephining in such cases. With the safe surgery of these days, this operation is performed with a

[1] See, *inter alia*, von Bergmann, "Die chirurgische Behandlung von Hirnkrankheiten," 3te Auflage, Berlin (1899), S. 389.

minimum of danger, and it has been tried many times for the relief of idiopathic epilepsy. It sometimes succeeds in arresting the fits for a time, as indeed the counter-irritation of any major operation may do,[1] but it is but very rarely that a permanent cure results. I can, however, recall one case of this kind in which trephining was successful. Nevertheless in idiopathic cases the operation can only be considered as a *dernier resort.*

Symptomatic epilepsy.

We pass now from idiopathic epilepsy to consider the symptomatic form—that is, those cases in which complete or partial epileptic seizures occur as the result of distinct and localised pathological changes in the cranial bones or within the cranial cavity. Many of these cases result from nonpurulent encephalitis and the other causes—pre-natal, natal or post-natal—of infantile cerebral paralysis, from tumours involving the cortex, from localised thrombosis, etc., or from one of the many traumatic causes from which cortical irritation may spring.

Causes.

Treatment.

These cases of symptomatic epilepsy stand in marked contrast as regards treatment with the

[1] See a paper by J. Maclaren, "Cases illustrating effect of Peripheral Irritations in Epilepsy," *Ed. Med. Journal*, vol. xx. (1875), p. 618.

idiopathic cases we have been considering. In the latter bromide treatment is very fairly successful, while operative measures are of little avail. In symptomatic cases the exact opposite is the case. Bromides do not produce, as a rule, any lasting effect on the fits, while operative measures are often successful. Certainly bromide treatment should be tried, but I fear that you will find the action of these salts very uncertain. Sometimes the fits may be diminished in number and severity at first, and they may even be arrested for a time, but even while the bromide is still being taken they are apt to return, and they may then show considerable and increased severity. If this occurs, you will probably find that it is quite useless to increase the dose of the bromide beyond, say, 90 grains taken during each day.

Trephining. In the traumatic cases where there is reason to suspect depression of the bone, splintering of the inner table, blood-clot, etc., and in those in which the convulsions in their Jacksonian character point to the presence of a tumour, a cyst or a removable scar, the operation of trephining is indicated.

These fits of cortical or Jacksonian epilepsy

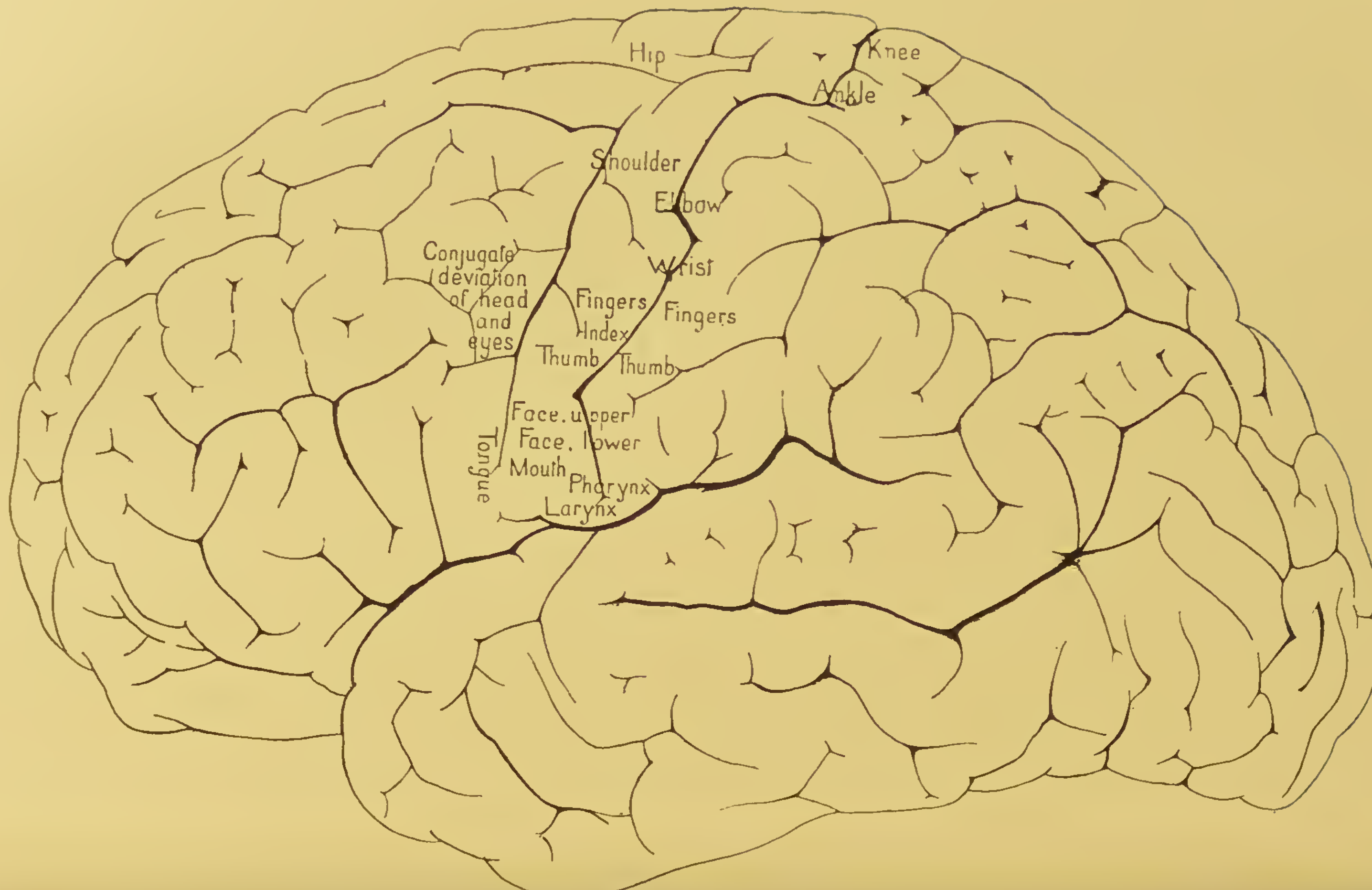
Jacksonian convulsions.

are very characteristic and of a high value in respect of diagnosis, and not less so in respect of treatment, especially in cases of tumour.

Their localising value.

The spasm usually begins in some special group of muscles, or even in one particular muscle, and subsequently, from radiation of irritation over the cortex to neighbouring centres, other muscle groups which these centres represent become involved in the convulsion. The exact way in which the spasm commences is indicative of the position of the cortical lesion and tells us where we should direct the surgeon to apply his trephine. If, for example, the spasm begins in the index finger and only extends subsequently to the other muscles of the limb, then the lesion may confidently be looked for over the centre for the index finger in the cortex on the opposite side of the body from that on which the spasm occurs. I give you a diagram showing the approximate position of these cortical motor centres.

It must, however, be remembered that the fits in these cases are not always of a Jacksonian character. A tumour lying on the motor cortex may, and often does, occasion a generalised convulsion. In some of these cases it may be



APPROXIMATE POSITION OF MOTOR AREAS ON LEFT CORTEX—*after* DEJERINE.

possible by careful observation to notice that some particular muscle group begins to twitch before the convulsion becomes general. In other patients you may find that the fit is sometimes Jacksonian and at other times general. All sorts of variations occur in these cases.

Results of trephining in traumatic epilepsy.

In cases of trephining for traumatic epilepsy it is surprising to find in what a small proportion of operations the results are lasting. The fits usually cease for a time, but are very apt to return after some months or in a year, necessitating a second operation which may or may not be successful. Braun [1] records a case in point, one of traumatic epilepsy, in which he had to operate three times, and only achieved permanent success on the third occasion, when he removed the cortical centre for the left arm, the spasm having always begun in the muscles of that limb. At the time the case was published the patient had remained free from fits for six years, so that the cure may be conceived to have been a permanent one. In these traumatic cases the prognosis as to the result of the operation of trephining depends largely on the position of

[1] H. Braun, "Ueber die Erfolge der operativen Behandlung der traumatischen Jackson'schen Epilepsie," *Deutsche Zeitschr. f. Chir.*, Bd. 48, p. 223.

the injury. When the bone has been injured over the motor cortex, trephining is usually successful provided the removal of the damaged cortex is sufficiently extensive. Trauma affecting those portions of the cortex lying posterior to the Rolandic area is a less favourable condition for operative interference; when the blow has fallen over the frontal cortex, operation is of little avail.[1]

Favourable and unfavourable cases.

Infantile convulsions.

In young children the upper motor neurone is much more sensitive to irritation than is the case in the adult. This is probably due, as Oppenheim suggests, to insufficient development of the mechanism of inhibition. Convulsions are therefore very common in childhood. In their phenomena these fits closely resemble the epileptic attacks of adults, but they have a much greater tendency to appear in series. Sometimes, of course, the fits of childhood are really the commencement of a true epilepsy, but more usually they depend on some more or less temporary cause, and when that cause is removed and the series of fits has ceased, the matter is at an end. By this I mean that the

[1] This question has been fully discussed by Victor Horsley, *Trans. of the Medical Society of London* (1903), p. 131.

nervous system of the child will not, as a rule, be injuriously affected, nor will its stability be impaired in the future by reason of these infantile convulsions.

It seems certain that, in the case of these convulsions, as in ordinary epilepsy, there is always present some predisposition, some inherited weakness of the upper motor neurone, which renders it more easily irritated. Certainly you must all have observed that children vary greatly in this respect, some having fits on slight occasion, others with greater cause showing no such explosions of cortical energy.

Predisposition.

But predisposition is not enough—there must also be an exciting cause; and, as in the epilepsy of adults, so in the eclampsia of childhood, that cause is usually some form of toxic action, some reflex irritation, or some organic brain mischief. The convulsions of childhood are common in the commencement of the various infective fevers, probably as the result of the action of the toxines of these maladies, but perhaps due directly to the increased temperature of the blood circulating in the cortex. Rickets has much to do with the causation of these fits in children, and this may be considered, with fair probability, to

Proximate causes.

be the result of auto-intoxication from the altered metabolic conditions of that disease. It need hardly be added that the poison of the uræmic state may, in children as in adults, irritate the cortex, and so cause convulsions.

Peripheral irritation.

When the cortex of the child is in that state of excitability which predisposes to such fits, peripheral irritation of almost any kind may bring on a series of convulsions. The more common of these are such irritations as that of teething, of gastric or intestinal catarrh, of the presence of intestinal worms, and even that of overloading the stomach with food may on occasion give rise to a fit.

Organic brain disease.

Various organic diseases of the brain or of its coverings may in children, as in adults, occasion epileptiform convulsions.

Treatment of infantile convulsions.

When we come to consider the treatment of fits in children, there is at once apparent a distinct difference as contrasted with the treatment of convulsions in the adult. In the latter no treatment is necessary or useful during the fit, except in cases of the status epilepticus. In the convulsions of children, on the other hand, it is possible and advisable to cut short the attack. This may be done by placing the

child in a warm bath, and if this does not suffice, by administering chloroform. The inhalation of this drug is a perfectly safe procedure in the case of a child, if carefully carried out. The irritability of the cortex may also be quieted by giving the child bromide of sodium, by the mouth if possible, if not, by the rectum. Chloral hydrate is also a most useful drug in these cases. It may be administered by the rectum in doses of five grains to a child of six months old, and of ten grains to a child of a year.[1] *Bromides.* *Chloral.*

At the same time it is necessary to get rid of any sources of peripheral irritation, and as these usually lie in the gastro-intestinal tract it is well to give an emetic and an enema. *Emetics.*

After the series of fits is over, it is advisable to give a little bromide for a day or two. Any evidence of the presence of rickets would suggest that the diet and general management of the child required readjusting.

Of tetany it may be said, with a near approach to certainty, that the phenomena which are characteristic of the disease are of toxic origin. Probably several forms of poison (toxines of *Tetany.*

[1] See John Thomson, "Guide to the Clinical Examination and Treatment of Sick Children," p. 136. Edinburgh, 1898.

infectious disease and of auto-intoxication) may so act as to produce the disease. Many considerations go to support this view. The disease sometimes occurs in epidemics, and when it is endemic it is seen much more frequently at some periods of the year than at others. It is also at times closely associated as a complication or sequela, with such diseases as measles and *Causation.* influenza. Curiously enough, shoemakers are specially liable to suffer from tetany, and possibly this may be due, as Oppenheim suggests, to exposure to some poison contained in the leather with which they work. That tetany occasionally arises from auto-intoxication is, I think, evident from the fact that it so often occurs in persons suffering from dilatation of the stomach. This question has already been discussed (see page 41) when we were considering the phenomena of gastro-intestinal auto-intoxication. That tetany has some curious relation to the function of the thyroid is evinced by the fact that the disease often follows the total extirpation of that gland, though it does not result from the operation if part of the thyroid be left.

The tetany of children is closely associated in many instances with rickets, and in others with

gastro-intestinal catarrh, and probably in both cases it is to be regarded as the result of auto-intoxication.

But while, from the above facts, it seems clear that we must look for the cause of tetany in some form of toxic action, there is no reliable or decisive evidence to show upon what set of neurones the poison acts. I cannot therefore say with any certainty that tetany is a disease of the upper motor neurone, and the treatment of the disease is considered in this lecture only as a matter of convenience.

Treatment of tetany.

The treatment of tetany resolves itself in the first instance into an attempt to remove the cause. Any gastro-intestinal catarrh, or other similar condition likely to give rise to poisoning, should be carefully treated, and suitable antiseptics given to prevent abnormal decomposition of the food. When the tetany has followed fever of any kind, an endeavour may be made to eliminate the toxine through the skin by causing profuse sweating. Pilocarpine administered with this object has sometimes proved useful. A more thorough means of accomplishing this object is to use the method of lavage of the tissues described on page 42. The casual relation between

Removal of toxines.

removal of the thyroid and tetany, to which allusion has already been made, suggests the advisability of thyroid feeding, and this has proved successful in some cases, though not in all.

Allaying the spasm.

The spasm may be allayed by the use of bromides, of chloral, of morphia, of hyoscine, and Hoche reports a favourable result from the employment of minute doses of curarine.[1] In the tetany of children the diet should be carefully regulated and any gastric or intestinal catarrh treated. In addition, the administration of phosphorus, iron and cod-liver oil is called for.

Chorea.

In speaking of chorea I do not refer to the post-hemiplegic form, the treatment of which is that of the hemiplegia which it has followed; nor to Huntingdon's chorea, but to the ordinary form, chorea minor, or Sydenham's chorea, popularly known as St. Vitus' dance.

Inherited tendency.

In common with so many of the diseases we have been considering chorea attacks those who inherit a predisposition to neurosis, and it appears chiefly in childhood and early youth; at a time, that is, when the power of cerebral inhibition is not fully developed. Such predisposing con-

[1] Hoche, "Versuche mit Curarin bei Tetanie," *Neurolog. Centralblatt*, 1894.

ditions being present, various exciting causes may precipitate the attack. Over-pressure in brain work at school may unquestionably start the attack, and the punishment which an ignorant schoolmaster sometimes inflicts on a boy, fidgeting in the early stage of chorea, may convert a mild into a very severe attack. Fright or other strong mental emotion may be the apparent starting-point of the disease. Pregnancy is also one of its causes, and in regard to this matter there are two points to be borne in mind: first, that these attacks occur chiefly in young women in their first pregnancy; and second, that it is much more common in unmarried women, in whom there is generally some mental distress and depression, than in the married.

Proximate causes.

Fright.

Pregnancy.

But while these and other similar conditions appear to precipitate the attack of chorea, there is, underlying it all, another cause, viz. toxic action. The virus which, in producing its effect on the nervous centres, gives rise to the phenomena of chorea, is probably, or perhaps one may say certainly, of different kinds. Occasionally, though rarely, the disease is seen as a consequence of scarlet fever or of measles, but by far its most frequent and most interest-

Toxic causes.

ing association is with acute rheumatism and acute endocarditis. That these affections are of microbic origin may now be regarded as established, and that it is to the toxine of these diseases that chorea is due can hardly be doubted in those cases in which they are associated. I think we may take it that the toxine in question is that of the diplococcus described by Poynton and Paine. In their experiments these observers found that inoculation of this microbe in rabbits not only gave rise to symptoms of rheumatism, but, in one instance at all events, produced nervous phenomena closely resembling those of chorea.

Association with rheumatism and endocarditis.

Pathology.

Assuming, as we may fairly do, that such poisons, and probably also those of auto-intoxication, may give rise to chorea, there remains to be determined on what parts of the central nervous system these toxic influences act. There are no constant morbid appearances to be found in cases of chorea when examined after death, but we may feel pretty sure that the action is an irritative one on the upper motor neurone, affecting, directly or indirectly, its cortical grey matter. We know, from the analogy of post-hemiplegic chorea, that lesions

of this neurone may occasion such symptoms. Monakow[1] argues, not to my mind very conclusively, that the lesion which gives rise to the chorea lies in the optic thalamus, and influences the upper motor neurone indirectly through centripetal fibres passing from the thalamus to the motor cortex. But whatsoever may be the site of the lesion in post-hemiplegic chorea, we may, I believe, hold that, in the disease we are now considering, the toxic action mainly affects the cortex, involving not merely the Rolandic area, but extending its effects over other portions of the cerebral grey matter. The sensory regions do not appear to be much affected; indeed alterations in the perception of the various centripetal impressions are the exception and not the rule in these cases, but those neurones which subserve mental operations are distinctly and often gravely involved.

Treatment.

If, then, in many cases at all events, chorea is the result of rheumatic toxine, can we in any way counteract the action of that poison? It is of course generally recognised that sodium salicylate has a powerful action on acute rheumatic

Sodium salicylate.

[1] Monakow, "Gehirnpathologie" in Nothnagel's *Specielle Pathologie und Therapie*, Wien (1897), Bd. 9, S. 330.

processes, and Lees[1] has recently strongly recommended the use of that drug in acute cases of chorea. So far as I have had an opportunity of testing it, his method of treatment has proved satisfactory. He gives the sodium salicylate in large doses, always in combination with twice the amount of sodium bicarbonate, which he believes aids its action. To a child of from six to ten years, a dose of 10 grains of salicylate may be given with 20 grains of the alkali. This should be administered every two hours during the day and every three hours during the night. Lees recommends that the doses should be increased gradually, if no untoward symptom manifests itself, until the quantity of salicylate reaches say 15 or even 20 grains, the sodium bicarbonate being correspondingly augmented. The total amount of salicylate which would thus be given in each 24 hours is, no doubt, very large, and great care should be exercised in watching the patient to see that no evil effects occur. This method of treatment is probably only suitable in cases in which the rheumatic toxine is at work.

Method of administration.

[1] Lees, "The pathology and treatment of chorea," *British Medical Journal*, August 29, 1903.

Passing from the question of treatment directed against the toxine itself, we come now to the consideration of the general measures which should be adopted in managing a case of chorea.

General measures.

It has been said that mental excitement may determine the onset of chorea in those otherwise liable, and we know that in attacks arising from other causes such excitement greatly aggravates the symptoms. It is therefore apparent how essential it must be that a patient suffering from chorea should be placed under conditions of rest and quietude. The motor neurones are excited and irritable, and the rest should therefore be bodily as well as mental.

Isolation.

When the diagnosis has been made, the child should be at once removed from school, and all lessons should cease. He should be placed in charge of a sensible nurse, capable of amusing and interesting him, and carefully isolated from his young companions. These, by noticing and calling his attention to his grimaces and odd movements, and commenting unfavourably on them, may do great harm to the nervous and quivering child. In many cases it is advisable to send the patient for isolation to a hospital

or a nursing home, particularly when his surroundings at home are not satisfactory.

Rest. Even in very mild cases it is best to keep the patient in bed for some days; indeed, the longer he stays there the better, if he does not become weary of bed and fretful. But it is often advisable to let a mild case be out of bed, for some hours at least, each day. The room he inhabits should be large and airy, and exposed as much as possible to sunhine.

Management of severe cases. But in cases which are at all serious, absolute confinement to bed should be insisted on. The child should lie on a soft mattress, or in severe cases on a water-bed, and the greatest care has to be taken to avoid the occurrence of bed-sores, by means of the precautions described in the last lecture. A patient, the subject of chorea, is specially liable to suffer from such sores, partly because the nutrition and resistance of the tissues are lowered, and partly as a result of the skin friction to which the constant jerkings give rise. In bad cases, also, the fæces and urine are apt to be passed in bed without due notice, and this increases the tendency to the formation of bed-sores.

When the muscular jerkings are so severe as

to threaten danger of bodily injury from trauma, it is well to place the bedding on the floor, and to surround the child with pillows or mattresses placed upright, so as to prevent contusion.

Hydro-therapeutic measures. In the very mild cases douching with cool water has sometimes a good effect. In all but the very severe, warm baths may be used. They have a soothing influence and seem to afford relief. Electrical treatment does no good, and its use is not advisable.

Arsenic. The remedy which is most frequently used in chorea is arsenic, and its value in many cases appears to be beyond doubt. The nature of its action is not known, but it seems probable that it controls in some way the action or the forma- *Its mode of action.* tion of the toxine. There is, indeed, some reason to believe that it excites phagocytosis, exerting a positive chemiotaxis in relation specially to the large mononuclear leucocytes. Be that as it may, there can be no doubt that it benefits the neurone and favourably affects the progress of the case. Some observations go to show that arsenic replaces phosphorus in the cortical neurotic cells, and if this is so, it may explain that diminution in the excitability of the neurone which follows its admininistration in chorea. It is usual to

commence with a small dose of Fowler's solution, say three drops, and to increase the quantity gradually up to eight or ten drops in the case of young children, and fifteen in older children and adults. This dose should be given three times a day, after food, the physician watching carefully for any sign of arsenical poisoning. In those cases in which, from gastro-intestinal irritation, the drug cannot be borne when administered by the mouth, it may have to be given hypodermically. Of late, certain other preparations of arsenic have been employed, such as the cacodylates and that di-sodic methyl-arsinate known as arrhenal. Of the use of these in chorea, I have, however, no experience.

Dose.

As for the other drugs which have been given for the cure of chorea, I have not much good to say. Nitrate of silver, oxide of zinc, sulphate of copper, which have sometimes been employed, are not to be recommended. In the hands of some, large doses of quinine have proved useful, but I have no experience of this method of treatment.

Other remedies.

Sedatives.

Various sedatives may have to be given in severe cases to control the jerking and to induce sleep. Of these, the bromide salts are of little

service in cases of chorea. Antipyrine does better and is well borne even by young children. But the most useful sedative is chloral. Its employment is not necessary in mild or even in moderately severe cases, but when the jerkings are so pronounced as sensibly to exhaust the patient, and particularly when sleep is much interfered with, the use of chloral is called for, and it then usually does well. The drug requires to be given continuously, and a careful watch should be kept on the state of the heart during its administration. The use of morphia is not to be recommended save in very severe cases in which chloral, given alone, has failed. The combination of a small dose of morphia with the chloral may then succeed in procuring rest.

Antipyrine.

Chloral.

During the course of the treatment the administration of tonics may be advisable. When the gastric functions are in good order, cod-liver oil should be given, and when there is anæmia it is well to order some preparation of iron.

Tonics.

In the stage of convalescence the power of control of the upper motor neurone over the muscles may be restored more quickly by means of regulated exercises similar to those employed

Regulated movements.

in locomotor ataxia for the re-education of the centres concerned in such movements.

In cases of the chorea of pregnancy, when the condition is severe, the induction of premature labour may be advisable.

LECTURE VII

ON THE TREATMENT OF LESIONS OF SENSORY NEURONES

The various centripetal impressions—Path of the sensation of pain—Causation of pain—Mode of action of remedies in stilling pain—Counter-irritation—Neuralgia—Treatment of trigeminal neuralgia—Treatment of sciatica—Treatment of the lightning pains of tabes—Migraine, its causation and treatment.

Difficulties of the subject.

We have now completed our study of the treatment of those diseases which mainly involve the motor tracts, and we pass to the consideration of those in which the lesion affects chiefly the paths conveying impressions from the periphere to the centres. We are here confronted with difficulties essentially greater than those we have already faced, for our knowledge of the mechanism and arrangements of the centripetal neurones is much less complete than is the case with regard to the motor tracts. These difficulties depend on various causes. The clinical

investigation of sensory affections, resting as it does so largely on the statements of the patient, must always be less exact than that of motor disorders. The difficulties in the way of experiments on animals are also much greater in the case of the centripetal tracts than in that of the centrifugal; indeed in many directions these difficulties seen almost insuperable. Finally, our knowledge of the mode in which drugs act on the centripetal apparatus is very imperfect, and this tends to make our treatment empirical.

Bearing these difficulties in mind, let us nevertheless make an attempt to consider systematically the treatment of what are commonly called sensory affections.

That term is a misleading one, for some important centripetal impressions never rise to consciousness in the individual, and only attract his notice when they lessen in intensity or accuracy, and still more when they fail.

Varieties of centripetal impressions. For present purposes we may classify the various centripetal impressions as follows:—

1. Tactual sensations.
2. Sensations of heat.
3. Sensations of cold.
4. Sensations of pain and various paræsthesiæ.

5. Muscular sense and the mechanism of co-ordination and equilibration.

Of these, the last forms so large, complicated and important a subject, that its consideration must be separately undertaken in a future lecture. And, so far as treatment is concerned, of the first four, the question of pain is by far the most important. To that we shall now devote our attention. *Sensation of pain.*

The end-organs for cutaneous pain consist, in all likelihood, of certain fine nerve filaments lying in the skin. From these the peripheral neurone for pain starts. Its fibres, lying in the peripheral nerves, pass through the ganglion on the posterior root (in the cells of which its trophic centre probably lies) and so enter the spinal cord, traversing the white matter and ending in the grey substance of the posterior horn. *Peripheral neurones.*

Up to this point our knowledge of the conduction-path of pain is tolerably certain. We know that destruction of the grey matter of the posterior horn abolishes pain over the skin-field corresponding to that segment. This knowledge is derived in part from experiment on animals. If the abdominal aorta just below the renal *Path of pain.* *Experimental investigations.*

arteries be compressed for about an hour there results necrosis of the grey matter in the lumbar region of the cord, with consequent sensory and motor paralysis of the legs.

That an acute anæmia of the cord could be produced in this way has long been known, and Stenson's experiment has been practised for many years in our laboratories. But as a means of investigating the paths of conduction in the cord this method appears to have been first used in 1884 by Ehrlich and Brieger.[1]

Results of ligature of aorta.

Since that date many researches have been made by this means,[2] and recently the resulting conditions have been investigated afresh by Loewenthal.[3] There can now be little doubt that such ligature of the abdominal aorta, while

[1] Ehrlich and Brieger, "Ueber die Ausschaltung des Lendenmarkgrau," *Zeitschrift f. klin. Med.*, Supplement zum Bd. 7 (1884), S. 155.

[2] The literature of the subject will be found in the following papers:—

Münzer and Wiener, "Beiträge zur Analyse der Function der Rückenmarkshinterstränge," *Neurol. Centralbl.* (1899), S. 962.

Rothmann, "Ueber die secundären Degenerationen nach Ausschaltung des Sacral- und Lendenmarkgrau durch Rückenmarksembolie," *Arch. f. Anat. und Phys.* (1899) (Phys. Abt.), S. 120.

Bochenek, "Dégénérescence des fibres endogènes ascendantes de la moelle après ligature de l'aorte abdominale," *Le Névraxe*, vol. iii. août 1901.

[3] Loewenthal, "Experiments on the conductivity of the spinal cord rendered anæmic by compression of the aorta," *Brain*, vol. xxv. (1902) p. 274, b.

causing acute necrosis of the grey matter of the lumbar and sacral cord, leaves the cells of the spinal ganglia and the root fibres untouched. The motor paralysis which follows is clearly the result of destruction of the anterior horns. The abolition of pain is due to the corresponding destruction of the posterior horns. The myelopetal fibres of the posterior roots conducting pain end in the grey matter of the posterior horn on the same side of the cord, and, consequently, destruction there breaks the connections of the path of pain.

Further proof in this direction is furnished by experiments in which the grey matter was destroyed directly by the injection of physiological salt-solution into the posterior horn.[1] In these cases the sensation of pain was abolished over that area of skin which corresponded to the segment involved.

Observations in syringomyelia.

The clinical phenomena met with in certain spinal lesions, particularly in cases of syringomyelia, strongly confirm this view. When, for

[1] Goldscheider and Flatau, "Ueber Hämatomyelie," *Zeit. f. klin. Med.*, Bd. 31 (1897), S. 175.

Münzer and Wiener, *loc. cit.*

Lubouschine, "Contribution à l'étude des fibres endogènes du cordon antéro-latéral de la moelle cervicale," *Le Névraxe*, vol. iii. p. 123, août 1901.

example, one posterior horn has been destroyed in the course of that disease, the sensation of pain, and also those of heat and of cold, are abolished over the corresponding segmental skin-field.

There can therefore be no doubt that the path of pain passes through the grey matter of the posterior horn. And since section of the posterior roots produces no degeneration which, traversing the grey matter, passes upwards, it is clear that the peripheral neurone for pain-conduction ends in that grey matter, and that the second neurone commences there. *Course of fibres conveying pain.* Clinical and experimental evidence alike show that the path leaves the grey matter almost at once and enters the white. But we do not yet know with certainty in what part of the white matter these endogenous fibres lie. Most probably they pass up the ventro-lateral columns, possibly in association with the ventral-cerebellar tract. Certainly some fibres in these tracts degenerate (as Lubouschine has shown) as a result of experimental lesions of the posterior horn on one side. There is indeed evidence to suggest that the pain path is a bilateral one, the heterolateral fibres crossing in the anterior commissure of the cord.

As to the further course of the path of pain

there is still greater obscurity. It seems likely, that the endogenous fibres arising from the posterior horns pass upwards as far as the basal ganglia, and that a synapse occurs in the corpora quadrigemina and also in the optic thalamus. Indeed, the comparative slowness of pain-conduction makes one think that the path of pain must be composed of many neurones. On these points, however, the greatest uncertainty prevails, nor do we know what part of the cerebrum is concerned in the perception of painful impressions, though some evidence points to the gyrus fornicatus as being its seat.

Causation of pain.

When we come to consider the causation of pain, as that symptom meets us clinically, we find that it arises in two essentially different ways. In the first place, the pathological process may be one affecting the peripheral sensory fibres for conducting painful impressions, or their end-organs either in the skin or in the deeper structures. This process is probably in most cases one of intense stimulation, but it may be that in some the peripheral nervous apparatus has become so unduly sensitive that stimuli of a physiological nature and degree are perceived as pain.

Peripheral stimulation.

In the second place, pain meets us clinically in cases in which, as the result of disease, the cortical perceptive centre has become so sensitive that physiological stimuli, either in the centre or reaching it through the centripetal paths, give rise to pain. This latter condition, known to many as pain hallucination, is common in hysterical and other so-called functional neuroses. In certain abnormal mental states, indeed, the disturbance of function may go further, and the sensation of pain may merge in, or even be overwhelmed by, that of pleasure. But to a lesser degree, even in health, the variations of pain perception are apparent to all of us. A blow or bruise received during a period of great mental excitement gives rise for the moment to no pain, whereas when the mind is unoccupied, and particularly when it is working introspectively, pain is keenly felt. But the consideration of functional pain or pain hallucination is not to detain us here. It will be discussed subsequently, in relation to hysteria and neurasthenia.

Pain from cortical causes.

What we have to consider now is that variety of pain which is truly of organic origin, arising either in the fibres of the peripheral neurone or its end-organs. This neurone stretches, as has

Peripheral causation.

been already said, from the periphere through the sensory nerves to the grey matter of the posterior horn of the spinal cord. It is at or between these two points that we must look for the lesion which gives rise to organic pain. For it is a very singular thing that, speaking generally (I shall mention the exceptions in a moment), organic pain does not arise from any lesion of the path of pain above this point. You may, for example, think of a case of intracranial neoplasm. In such a patient no pain arises except such as is due to rapid increase of intracranial pressure, to involvement of the membranes, or to pressure on sensory cranial nerves, and in these cases it results from stimulation of peripheral neurones. Even in cases, for example, in which a hæmorrhage has torn up the posterior part of the internal capsule, no pain need result, though the total anæsthesia on one side of the body shows that the path of pain has been involved. Indeed, it may be held that lesions of the cerebral substance itself are painless. The same may be said of the path of pain in the spinal cord, from the grey matter of the posterior horns upwards. A myelitic process, for example, may advance so as to destroy the endogenous fibres conducting pain, and thereby

Peculiarities as to point of origin of pain.

produce analgesia, without there being an early stage during which pain is felt. In a process of this kind one would expect that the inflammatory irritation which precedes destruction would give rise to acute suffering. But this is not the case except in so far as the peripheral neurones of the segment in question may themselves be involved.

It would thus appear that before a stimulus which is proceeding up the path of pain can give rise to a painful sensation, it must have some quality imparted to it by the peripheral neurone in order that it may be recognised as pain by the cortical perceptive mechanism. What this impress is, it is impossible at present to say.

"Central pain."

This then is the general rule. There are, however, as has been already said, certain exceptions. A few cases are on record in which severe pain has been felt as the result of cerebral lesions. Edinger,[1] for example, has recorded instances in which this symptom followed a lesion in the optic thalamus. Another example of a similar lesion followed by similar symptoms has been described by Schupfer.[2] There are a few

[1] Edinger, "Giebt es central entstehende Schmerzen?" *Deut. Zeit. f. Nervenheilkunde*, Bd. 1 (1891) S., 262.

[2] Schupfer, "Sui dolori di origine centrale," *Rivista sperimentale*

others scattered through the literature. But the very rarity of such cases suggests that the cause of the pain was of some other origin than interference with the pain path. It is to be remembered that the optic thalamus stands in intimate relation to the mimetic movements, as of laughing and crying, and that it lies in very close association with the cortex. Its relation to certain post-hemiplegic phenomena, particularly to athetosis, is well known, and as this is probably due to a reflex through centripetal fibres to the motor cortex and thence down the motor tract, it may well be that the pain which sometimes though rarely follows thalamic lesions may also be of reflex origin.

Stilling of pain.

But while, as we have seen, the various kinds of organic pain with which we have now to deal may, for all practical purposes, be considered as due to changes in the peripheral neurone, and not to more central lesions involving the conduction path, the stilling of pain may be accomplished by influences acting at any level in that tract. Whether the peripheral end-organs are paralysed, or the fibres of the peripheral neurone,

di Freniatria, vol. xxiv. (1898), p. 582. The literature on this interesting subject will be found in this article.

or the synapse in the grey matter of the posterior horn, or the endogenous fibres running up the cord, or the conduction path in the brain, or the cortex, the result is the same. Pain ceases to be felt.

Action of remedies.

The great difficulty in discussing the subject of treatment in relation to pain lies in the fact that with few exceptions we do not know at what point or points in this long linkage of neurones any particular remedy acts. No doubt our knowledge of the action of the local anæsthetics is fairly accurate. As to the local action of cocaine, for example, we know that after a short period of slight irritation, it paralyses the peripheral apparatus of touch and of pain, and that it also affects the perception of heat and of cold. Carbolic acid, locally applied, acts much in the same way. We know also that menthol applied to the skin stimulates the end-organs connected with the cold sensation, and subsequently produces some anæsthesia. We may also fairly assume that cocaine when injected into the spinal sac produces its anæsthetic action by paralysing the fibres of the peripheral neurone in the posterior roots as they enter the cord.

The general anæsthetics paralyse the sensory apparatus in the cortex and in this way abolish pain, but it is by no means clear that they do not act also on some of the neurones conveying pain. This is indeed highly likely, and the dissociation of sensation sometimes seen during ether narcosis points strongly in that direction. In how far the pain-stilling action, produced by such drugs, may be attributed to interference with the amœbism of the protoplasmic processes of sensory cells, it is impossible to say with certainty. All that can safely be stated is that the results of experimental enquiry point to some extent in this direction, and that such a theory is not at variance with clinical observation. *General anæsthetics.* *Amœbism.*

The whole subject of the action of drugs in subduing pain is indeed very obscure, and it seems clear that the only way in which the question now before us can be profitably discussed is to describe the methods of treatment to be adopted in each of those diseases in which pain is a prominent symptom. *Obscurity of the subject.*

But before proceeding to consider in detail the treatment of these various sensory disorders, it may be useful to say a word as to that method

Counter-irritation. of treatment usually known as "counter-irritation." You are of course aware that in many cases a deep seated pain may be relieved by some application which irritates some particular skin area, and you have often seen how satisfactorily a small blister will remove the pain of pleurisy. But while counter-irritation acts exceedingly well in cases where pain is the result of visceral disease, I feel bound to tell you that it is not, as a general rule at least, so satisfactory a method of treatment in those cases in which pain occurs in the course of nervous disease.

Its theory. The theory of counter-irritation and of the good effects in relieving pain which follow its use, depends almost certainly on that very curious correlation of the afferent visceral and afferent somatic nerves to which is due the phenomenon known as "referred pain." It is *Head's work.* almost entirely to the beautiful work of Head that we owe our knowledge regarding this interesting point.

As you know, each viscus has a special relation to a particular area of skin corresponding to the spinal segment with which the viscus in question stands in nervous connection. If

the viscus is diseased in such fashion as to originate a centripetal impression, this irritation somehow radiates in such a way as to produce stimulation in the nerve fibres coming from the corresponding segmental skin area, and so gives rise to what we call "referred pain." The afferent visceral nerve fibres and the afferent fibres from the skin come into near relation in the ganglion on the posterior spinal root; but whether the radiation takes place there, or in the grey matter of the cord is not certainly known.

Converse position.

And just as disease of the viscus causes referred pain or referred hyperalgesia in the corresponding segmental skin area, so irritation of this skin area (as by a blister) often acts very beneficially on the visceral disease. This, however, does not specially concern us here. What is of interest to us is rather the converse—a consideration, namely, of how somatic segmental pain may be lessened or removed by acting on the viscera. Just as pain and hyperæsthesia of the scalp are often produced by disorders of the abdominal organs, so a prompt action on the gastro-intestinal tract may remove the pain of headache. And a similar consideration may so far

explain the phenomenon in those cases of tabes in which the lightning pains are relieved by prompt evacuation of the rectum by means of a purge. The lower bowel is thus stimulated, and the centripetal impulse passes to the sacral segments, where also the nerve fibres corresponding to the lightning pains pass. One may suppose that some species of interference takes place between the two sets of nerve impulses, visceral and somatic, either in the posterior root ganglion or in the grey matter of the segment, and that thus the lightning pains are benefited.

Let us now pass to the consideration of the treatment of some of these diseases in which pain is a prominent symptom.

Neuralgia. Pain meets us very frequently in practice in that disease or set of diseases which we know as *neuralgia*. Properly speaking we ought to understand by that term a condition in which paroxysmal pain, referred to the area of distribution of a nerve, is the main symptom, and in which no evident organic changes are present in the sensory neurone involved. This definition is indeed true in many cases, for a severe trigeminal

neuralgia may go on for years and yet the most careful microscopic examination after death may fail to show any changes in the fifth nerve, or in the Gasserian ganglion. But, in a considerable number of instances, structural degenerative changes do arise, and the pain in such cases may be rather that of neuritis than of true neuralgia. The clinical distinction between these two processes—the one inflammatory or degenerative, the other purely functional—is not easily drawn. In many of the cases of neuralgic pain which you will be called on to treat, the pain is purely symptomatic of a gross lesion affecting the sensory neurone in question. For purposes of treatment, however, we may conveniently group these forms together.

Treatment of neuralgia.

The consideration of the treatment of neuralgia is of great practical importance for the following reasons. The condition meets one frequently in ordinary practice. The pain is so severe as to call urgently for relief. The treatment is often difficult, and sometimes the obstacles are almost if not quite insuperable. And, in bad cases, when you see the physique of the man breaking down and his morale giving way under the severity of agonising pain you

will realise how every energy you have must be strained to find means of relief.

Causal indications.

Clearly, it is most important to discover the cause. But, in any individual case, this is not always possible. Nor, when you do detect the cause, is it always a removable one. And even if you are so fortunate as to trace a definite cause, and so happy as to be able to remove it, there are instances in which the paroxysmal neuralgic pain still continues. You may compare this with those cases of epilepsy, sometimes encountered, in which removal of the original cause does not immediately stop the fits, and in which we may suppose that the cortex, having got into the habit of explosive discharge from time to time, cannot readily revert to a reasonable behaviour. As a rule, however, in neuralgia removal of the cause tends to cessation of the pain.

When you are called to see a case of severe neuralgia, you will find that the patient will not give you time to hunt for the real cause of the condition. He expects you to give him relief at once. This is natural and right. How such immediate relief may best be obtained we shall see presently. In the meantime let us consider

the causes which underlie, promote, and actively give rise to the neuralgic condition.

Trigeminal neuralgia

Let us take, in the first place, *trigeminal neuralgia*, for neuralgia of the fifth nerve is common; it is the severest form of the disease; and its treatment is often very difficult.

Its causation.

When you come to consider the causes which underlie this disease, you will find that in a very large proportion of cases there is evidence of neurotic inheritance. An investigation of the family history will show that parents or other near relatives have suffered from neuralgia or from some other of that group of diseases which we find so often in neurotic families. Indeed the patient himself may be the subject of neurasthenia as well as the victim of neuralgia. On the weakened neurones of an individual of such heredity any nerve-strain tells acutely. In women at the time of puberty, during pregnancy, at the menopause, such strain comes, and it is then that neuralgia is apt to show itself. In the same way strong emotion may precipitate an attack in those predisposed, as may also any considerable muscular strain. Sexual excesses, likewise, may form the starting-point of neuralgia, and although in many such cases the pain may limit

itself to the testes and neighbouring nerve areas, in others the strain tells in such a way as to produce neuralgia in more distant regions, including that of the fifth nerve. The indications for treatment which these conditions present have been sufficiently considered in a previous lecture.

Anæmia. Malnutrition is also a potent cause, and in anæmia the true source of the neuralgia with which we are dealing often discovers itself. The use of iron is then called for, and in most cases the best preparation you can give is that known as Blaud's pill. Capsules containing a quantity of the pill mass equal to three pills are usually readily borne, and I am in the habit of giving one of these capsules in the day, for three or four days, then two, and ultimately three. It has seldom appeared to me to be needful or desirable to increase the dose beyond this point. But you will occasionally come across a case in which, on account of gastric disturbance, iron cannot be so administered. Under such circumstances I have had most successful results from giving the iron hypodermically. As you know, when iron is administered by the mouth, even in the best form, a great deal is not absorbed but is excreted

Administration of iron hypodermically.

by the bowel. For this reason, if you give the iron hypodermically, you will find that a much smaller dose is necessary, and this is a fortunate circumstance, for the preparation of a solution for hypodermic use is not easy. The preparation[1] I employ is one of pure ferric citrate dissolved in distilled water in such a proportion that ·07 gramme is contained in 2 c.c. The quantity necessary for each single injection is placed in a glass capsule, one end of which is finely drawn out. It is then carefully sterilised by heat, and the tube closed by fusing the glass. When you are going to use it, you break the glass point, introduce the needle of your syringe and suck up the solution contained in the tube. The injection ought to be made into muscular fibre, and as a rule little or no irritation follows. It is usually sufficient to give such an injection twice or thrice in each week.

But in neuralgia resulting from anæmia, iron is not the only remedy you should employ. It is very desirable, in many cases, to give arsenic also. Blaud's capsules are sold containing $\frac{1}{60}$ grain of arsenious acid, and you will find these *Arsenic.*

[1] This solution, in such capsules, has been very carefully prepared for me by Mr. George Lunan, Pharmaceutical Chemist, Queensferry Street, Edinburgh.

excellent in such cases. If you have to give the iron subcutaneously, you may usefully combine with it a minute dose of arsenic or of strychnine.

But it is not merely in such cases of general anæmia that neuralgia results. It seems probable that the trigeminal neuralgia one sees in oldish persons, the subjects of pronounced arterio-sclerosis, may result, to some extent at least, from defective local circulation in the nervous tissues involved. Such cases are apt to be very

Iodide of potassium. intractable and demand careful consideration and treatment. You will, I think, get most good from a combination of iodide of potassium with

Nitro-glycerine. strophanthus. Or you may sometimes usefully substitute liquor trinitrini for the iodide. Some people use digitalis, but I consider strophanthus better in these circumstances. Of course the diet requires careful regulation in all such cases of arterio-sclerosis.

Very frequently you will find that the neuralgia with which you are dealing depends on

Toxines. the presence of some toxine. Alcohol, tobacco, lead and other such poisons may bring it about, and in these instances your line of treatment is apparent.

Or again the poison acting may be one of that

group which we include under the term "auto-intoxication." Very frequently the absorption has taken place from the intestines and no one can be in practice for long without recognising how often neuralgic pain is associated with and dependent on a state of constipation. A regular and sufficient action of the bowels should therefore be procured in all cases, and you will find that a combination of aloin, extract of belladonna, and extract of nux vomica is very useful for this purpose.

Auto-intoxication.

But constipation is not the only thing to be attended to in this connection. If there is reason to think that abnormal decomposition and fermentation are going on in the digestive tract, with consequent auto-intoxication, it is well to limit these processes. This you will best do by giving regular doses of some preparation of mercury along with salicylate of phenol or salol. I usually give a quarter of a grain of grey powder, or $\frac{1}{12}$ grain of calomel, with 8 grains, or more, of salol in a cachet three or four times a day. The salol requires to be finely divided if it is to act properly, and this may be secured by seeing that the powder is well rubbed down before it is placed in the cachet. Or, what I think is still

Antiseptic remedies.

better, the finely powdered salol may be levigated and made up as a mixture in water, held in suspension by means of tragacanth. In this case I give a smaller quantity of salol, say 3 or 4 grains. A minute dose of perchloride of mercury ($\frac{1}{60}$ or $\frac{1}{30}$ grain) may usefully be administered, not along with the salol, but at a different time of day.

In some such cases ichthyol appears to do well. It may be given in doses of 5 grains or so, dissolved in water to which a drop or two of oil of verbena has been added to cover the taste. In the same solution you may give a minute dose of perchloride of mercury.

Gout. The metabolic changes which occur in gout frequently give rise to neuralgic conditions of greater or less severity. In such cases the primary malady must be attacked. I think nothing acts so well as a combination of quinine, salicylate of soda and colchicum. The wine of colchicum may be used, but of late I have seen excellent results from the administration of colchicine.

Diabetes. The neuralgia which is sometimes met with in cases of diabetes belongs to the same class. In it also the main thing is to treat the disease in the

course of which it has occurred. The pain may be subdued for the time by means of such remedies as salicylate of soda, aspirin and phenacetin.

Often, however, the toxines which give rise to neuralgia are those which are formed in the course of infective diseases. *Malaria.* Malaria is a potent cause, affecting chiefly and characteristically the first division of the fifth nerve. Here quinine in large doses is required. *Influenza.* Influenza frequently enough gives rise to neuralgia, and here, too, the area of distribution of the first division of the fifth nerve is apt to be the seat of pain. In this case, as well as in those in which rheumatism is the cause, and in the cases which find their starting-point in exposure to cold, salicylate of soda, sulphate of quinine, and phenacetin is in my experience the combination which gives the best results.

New growths. New growths and other processes causing pressure on the nerve are apt to give rise to symptoms of neuralgia, and these have to be dealt with according to the circumstances of the individual case. In connection with the fifth nerve, pressure is very apt to occur in the bony canals before the branches emerge on the face. *Syphilis.* Syphilitic changes in the bone and in the peri-

osteum are the commonest cause of this, and when trigeminal neuralgia arises in the course of that disease, anti-syphilitic treatment by means of mercurial inunction and iodide of potassium is urgently called for. The details of this treatment have been considered in a former lecture.

Trigeminal neuralgia is very frequently excited by some irritation in connection with the tissues to which the branches of that nerve are distributed. Thus, diseased processes within the orbit may excite neuralgia, as may also the great ocular tension which occurs in glaucoma. But less disturbance than this may be the exciting cause of much pain. Errors of refraction, when they are uncompensated, may do so; and this is especially the case with regard to astigmatism. I have repeatedly seen a quite trifling degree of astigmatism give rise to much pain—a pain which at once subsided when the error was corrected by proper lenses. In the same way affections of the middle ear or of the mastoid may be the starting-point of neuralgia, and so may diseased conditions of the nose and the various sinuses. But probably a still more frequent cause is to be found in connection with the mouth. Diseases of the tongue, of the alveolar processes, and

Ocular conditions.

Ear conditions.

especially of the teeth, are often the immediate cause of severe facial neuralgia.

When, therefore, you are confronted with a case of trigeminal neuralgia, it is proper that you should carefully assure yourselves that there is no morbid condition present in any of these situations which might reasonably be supposed to be the exciting cause of the neuralgia. This search should be a careful one, but it may easily be overdone, and too much importance may be attached to such a possible cause of the malady. One often sees cases in which healthy teeth have been profusely and needlessly sacrificed to the enthusiasm of the practitioner, without in any way reducing the severity of the *tic douloureux*, to cure which they were removed. You will not, therefore, at once proceed to render your patient edentulous because he happens to have some facial neuralgia. If, however, a tooth is carious, it must be looked to, and if on tapping an apparently healthy tooth the neuralgic paroxysm is at once started, that tooth had better be removed.

Irritation from diseased teeth.

But while in many cases you will be able to find the cause of the neuralgia, and will, in this light, direct your treatment to a successful issue,

there remains a certain number of cases in which this knowledge is not attainable; and it is an unfortunate thing that those cases in which no evident cause is discoverable are very often the severest of all, the genuine *tic douloureux*. In such patients during the attack there is apt to be spasm of the facial muscles, and Trousseau used to call this condition epileptiform neuralgia.

Epileptiform neuralgia.

In this affection there is usually a well-marked neurotic heredity, accompanied with malnutrition, and it is in them that the mental functions are so apt to become affected and the whole morale shattered. This, and the agonising pain which occasions it, lead sometimes to actual suicide, often enough to the contemplation of that act. The malnutrition in such cases is largely due to the fact that the act of chewing brings on the paroxysm. The feeding, therefore, requires to be very carefully attended to. As a rule in such severe cases the milder sedatives are useless, and one is driven to use morphia. And it is in them that the resort to surgical aid sometimes becomes ultimately necessary. We shall consider these points presently.

In all cases of neuralgia the question of

nutrition is an exceedingly important one. The better fed your patient is, the more likely is it that he will throw off the malady, always provided that his power of assimilation is good, and that excretion is going on satisfactorily. In ordinary cases it is probably sufficient to see that the patient is taking a fairly liberal mixed diet. But when the case is a severe and difficult one, it is well to consider whether the Weir-Mitchell treatment ought not to be adopted. The regular overfeeding and the accompanying general massage do well in some cases. It need hardly be added that the administration of cod-liver oil is often very beneficial.

Feeding of such cases.

Then again there is the question of climate to be considered. In obstinate cases and where the patient can afford to go abroad, it is quite worth while to try the effect of residence for a time in a warm and dry climate.

Climate.

Turning now to the immediate treatment of the attack itself, you will remember that I told you that in practice one has usually to employ means to still the pain before proceeding to that careful enquiry which is usually necessary before its cause can be made clear. Now, while in

Treatment of neuralgic attack.

many cases a resort to morphia will ultimately prove to be necessary, it is always well to delay this as long as possible. Such a disease as neuralgia, with its recurring attacks and in the severity of its sufferings, presents just those conditions which favour the acquiring of the morphia habit, and this the more in that the patient so often shows a neurotic inheritance.

Analgesics. Therefore, when you are called to see a case of neuralgia—and we are specially considering trigeminal neuralgia just now—you will first of all try to soothe the pain by means of the milder analgesics. You are indeed embarrassed by the number and variety of such drugs which lie to your hand. A modest computation has set the count as high as one hundred and fifty. Of these, of course, but few are really or widely serviceable.

Quinine. One of the best of these remedies is quinine. Its use is, of course, specially indicated in cases in which there has been malaria. These, however, we seldom see here. But even when all possibility of a previous malarial infection has been excluded, quinine is often of much service. I have frequently seen brilliant results from its administration in neuralgia of the fifth nerve—

particularly, I think, where the affection is one of the first division. I have usually employed the sulphate, and in large doses,—say of 10 grains,—which may be repeated two or three times until the pain subsides or the physiological action is thoroughly manifest.

Salicylate of soda is another valuable remedy *Salicylates.* in these cases, though not so powerful or so immediately effective as quinine. You may usefully combine phenacetin or antipyrine with it. *Antipyrine.* Indeed I have seen excellent results from a powder containing 5 grains each of sulphate of quinine, salicylate of soda and phenacetin. *Phenacetin.* The new preparation known as aspirin (acetosalicylic acid) has served me well in some cases. Given in quantities of from 5 to 10 or even 15 grains, it does not appear to produce unpleasant symptoms unless the dose is frequently repeated and at short intervals, and it certainly acts beneficially in some cases of neuralgia.

Exalgin at times suits well as an analgesic, though its action requires careful watching lest it produce collapse and cyanosis. It is well to begin with a small dose (2 grains), which may be gradually increased. Another remedy of the *Pyramidon.* same class is the substance known as pyramidon.

In the hands of some (notably Oppenheim) it has proved most useful, and it has sometimes served me well. It is a dimethyl-amido derivative of antipyrine, and in its action it resembles that drug. Its effects develop more slowly, but they are more lasting than is the case with antipyrine.

Butylchloral. Butylchloral hydrate is an excellent remedy in neuralgia, at any rate for the purpose of stilling the pain for the time. It may be given in doses of 5 to 10 grains, or even more, and is best administered in solution, with glycerine or syrup.

Gelsemium. The pain of facial neuralgia is sometimes wonderfully relieved by the use of gelsemium sempervirens. The usual preparation is the tincture (5-15 minims), but some prefer to give gelsemine hydrochloride, the dose of which may be said to be from $\frac{1}{200}$ to $\frac{1}{100}$ grain. Aconite may also be of service, and with some it is a very favourite remedy.

Iodide of potassium. Iodide of potassium in full doses is often very beneficial, and with it the bromide salt may usefully be combined. Bromides alone are not of much service in trigeminal neuralgia, save in those rare cases in which the neuralgic paroxysm replaces a true epileptic attack. Turpentine,

though it is sometimes of benefit in cases of trigeminal neuralgia, finds its most useful employment in sciatica.

Arsenic.

Allusion has already been made to arsenic. The importance of this remedy in such cases is great.

In what has been said I have endeavoured to mention the more important of those remedies for the relief of pain which are given in cases of facial neuralgia, and particularly those of which I myself have had experience. It is as a rule impossible, in any individual instance, to say what remedy will prove successful. One has to try one after another until the appropriate method of treatment is found, if indeed you are so happy as to discover it. And in regard to these drugs, the general remark may be made that they usually require to be pushed to rather full doses before the desired result is attained. How far it is safe to go depends on the strength of the patient and, of course, on the nature of the drug, but in most cases it is well to give some form of stimulant along with any drug of which the action is a depressing one.

Morphia.

But, after all, in too many instances you will find that a resort to morphia becomes necessary.

I am not by any means convinced that the hypodermic use of that drug is not of itself curative, as well as merely palliative. In any case it is a wonderful pain-reliever. But, for the reasons already mentioned, its employment should be delayed as long as is reasonable. The danger of starting a morphia habit is great, and especially for this reason the doses should always be administered by the physician, and the syringe never allowed to pass into the hands of the patient.

Its administration. If you have reason to believe, from past experience, that no idiosyncrasy as regards morphia exists in the case with which you are dealing, then I consider it best to commence with a full dose. This will relieve the pain for some hours, and so allow other remedies to have time to act. The addition of atropine to the hypodermic injection is often advisable. In bad cases such injections have to be repeated from time to time, and in the worst forms of *tic douloureux* the patient may have to be kept under morphia for long, so long indeed that even with large doses the relief obtained becomes only slight and evanescent. It is in such circumstances that surgical interference

becomes specially necessary. To this point I shall return presently.

Counter-irritation. Passing now to the local treatment of facial neuralgia, you will not, I think, get much benefit from the use of counter-irritation or of liniments, save in slighter cases. In these the liquid formed by the mixture of chloral hydrate, camphor and menthol (equal parts of each), may, when rubbed into the skin, give some relief. The application of cocaine solution to the conjunctiva or to the nasal mucous membrane is sometimes helpful.

Heat. The local application of heat, particularly of dry heat, is of great benefit in many cases. It is difficult to apply the heat radiated from electric lamps to the face, but in other forms of neuralgia I have seen great benefit follow such treatment.

Electricity. One other kind of local application, and that the most important, remains to be mentioned, viz. electricity. The use of the galvanic current often gives excellent results in cases of trigeminal neuralgia. The anode, or positive pole, should be applied over the seat of pain, and the negative should be placed on the sternum. Rather large electrodes should be employed and

the current should not exceed 1 or 2 milli-ampères. This form of treatment is specially useful when the case is treated early. Static electricity and the use of high frequency currents are also to be recommended in this condition. The relief of pain after such treatment is often very wonderful.

Osmic acid.

In obstinate cases osmic acid has been used on the recommendation of Hughes Bennett.[1] The nerve is exposed by incision, and a few drops of a 1 per cent solution of osmic acid are injected under its sheath by means of a sterilised hypodermic syringe. I have no experience of this method of treatment.

Surgical measures.

The last resort in severe and obstinate cases is to the knife of the surgeon. The wonderful advances in technique which surgery has made of late years have enabled much to be done with safety which could not previously be attempted, and have brought even so severe and difficult an operation as that of excision of the Gasserian ganglion within the sphere of practical therapeutics.

When, then, you encounter a case of trigeminal neuralgia which has resisted treatment,

[1] *Lancet*, 1899, vol. ii.

and in which the suffering is so severe as to render life miserable, it becomes a question whether surgical interference is not advisable or even necessary. Under what conditions are you to come to such a conclusion? I need hardly say that an opinion of that kind is not to be lightly arrived at.

Grounds on which surgical interference is called for.

In the first place, then, you ought to satisfy yourselves that all reasonable measures in the way of treatment have been tried, and tried thoroughly; that the remedies likely to do good have not only been given, but that they have been given in as full doses as are necessary to delevop their action distinctly. You should assure yourselves, further, that the neuralgia does not proceed from gout, rheumatism, diabetes, or other similar general disorder, and that it is not hysterical in its origin; in short, that there is no removable cause to be found, that the attack is a long continued one, and that it defies treatment. But, inasmuch as the disease sometimes lasts long and yet is completely recovered from without operation, you ought further to satisfy yourselves that the attacks are of such severity as seriously to undermine the health, mental and physical, of your patient. Finally you should

consider whether your patient has strength enough to stand the operation. Practically you will find that such cases are chiefly those known as epileptiform neuralgia.

Variety of operation. If then, in view of such considerations, you arrive at the conclusion that surgical interference is desirable, you have next to consider what variety of operation is most likely to suit the special case. Now, of all the forms of neuralgia met with in practice, that connected with the fifth nerve is the one which best lends itself to such measures, and many more operations have been performed on that nerve than on all the other nerves of the body. This is due to the frequency with which trigeminal neuralgia occurs, to its severity, and to the fact that the nerve is so largely sensory. A mixed nerve cannot be interfered with in the same way. If only one of the branches of the nerve is affected, say the first division, the simplest operation is that of section. But this is not one to be recommended, for though the pain is usually at once relieved when the nerve is divided, it returns as regeneration takes place. Neurectomy, in which a portion of the nerve (not less than 1 cm.) is cut out, is much more satisfactory, for it usually procures

a long period of relief, and it sometimes effects a complete cure. Or you may direct the surgeon to proceed further, and to perform the operation of avulsion, in which the nerve is extracted by force. This operation, combining as it does the mechanical effects of nerve stretching with neurectomy, is very often successful, and it is the one to be recommended. When the second division of the fifth is so operated on, Meckel's ganglion is usually torn out along with the nerve.

Excision of Gasserian ganglion.

In very severe cases in which these operations have failed, and in those in which all three divisions of the nerve are involved, it becomes necessary to consider whether the surgeon ought to proceed to an intracranial operation—that is, to excise the Gasserian ganglion or to divide the nerve fibres behind that point. Now, this is one of the most formidable operations in the whole range of surgery, and no surgeon who has not a wide and intimate experience of the technique of his craft ought to undertake it. The mortality is high—how high I cannot tell you, for the tendency, I am afraid, is not to report unsuccessful cases so frequently as those which succeed. Probably we shall not be far wrong if we consider

that of all the cases operated on between 20 and 30 per cent die.[1] Still, in a very severe case, even such an operation as this is sometimes advisable, provided the strength of the patient is fairly good. *High mortality.* He must of course be informed of the risks attending the operation, but such risks sink into insignificance in his eyes in view of the commanding pain from which he is suffering. The operation has frequently proved successful, and is certainly one the claims of which should be carefully weighed in all severe cases. The removal of the Gasserian ganglion has sometimes been followed by keratitis and destruction of the eyeball, but this does not appear to be a necessary result, and it can probably be avoided by due care and careful protection.

I shall not attempt to describe the technique of these various operations. It is sufficient to indicate, as has been done, the main points which should guide you in deciding for or against operative interference.

Sciatica. When we pass to the consideration of sciatica,

[1] No surgeon has operated on the Gasserian ganglion so frequently, or so successfully, as Krause. I find from his last statistics ("27 intra-kranielle Trigeminus-Resektionen, darunter 25 typische Exstirpationen des Ganglion Gasseri, und ihre Ergebnisse," *Münch. med. Woch.* (1901) S. 1043) that out of 27 operations 6 were fatal.

we find at once a marked difference as to the lines on which treatment is to proceed. This depends on various causes, largely on the situation and distribution of the nerve, but most importantly on the fact that so many of the cases of sciatica which present themselves for treatment are not instances of true neuralgia at all, but are in reality due to neuritis. In these you will notice that the exact style of the pain is not that characteristic of true neuralgia, and you will find other evidence, in the shape of muscular atrophy and the like, going to show that the symptoms are those of neuritis.

It is therefore not a matter of surprise to find that of those cases of sciatica which come before us there are but few in which heredity plays any important *rôle*. Much more commonly the condition arises from mechanical causes or from toxic agency.

Thus, in commencing to treat a case of sciatica, the most important line of investigation is that fitted to disclose its cause, and this search will be more frequently rewarded with success in sciatica than in trigeminal neuralgia.

What then are the causes for which we are to search in this very common form of neuralgia? *Causation.*

Apart from aetual trauma, of the oceurrenee of whieh in all likelihood you will be at onee informed by your patient, you have to look for the presenee of anything whieh may eause pressure on the nerve.

Pressure outside pelvis.

Outside the pelvis the search is fairly easy. Any tumour or bony outgrowth may produce pressure suffieient to give rise to sciatica. And I need not say that you should search for sueh, and that you should not overlook the possible presenee of a neuroma, true or false. In treating such a case the aid of the surgeon is usually neeessary.

Pressure within pelvis.

Conditions inside the pelvis leading to interference with the nerve fibres before they unite to pass through the great saero-seiatic foramen are often less easy of deteetion. In a woman uterine conditions—normal or abnormal—have to be looked for. Tumours and phlegmonous exudations may likewise produee the required degree of pressure. But we need hardly delay to consider such questions here. More interesting to us are those cases of sciatica in whieh the eondition arises as a consequenee of venous engorgement in the neighbourhood of the strands of the plexus, from which the nerve takes its origin. Sueh congestion, which is most easily

set up in plethoric persons, is certainly in many cases the starting-point of sciatic pain. Now, this venous engorgement may arise in many ways. It may occur, for example, in cases of heart-failure and general backward pressure, but more frequently some hepatic condition is its cause. It is often associated with, and sometimes directly caused by, fæcal accumulation, and this is a point requiring very careful investigation in all cases of sciatica.

Venous engorgement.

If, then, you find evidence pointing to the presence of venous engorgement within the pelvis, what line of treatment are you to adopt? If there is some cardiac lesion present, and if compensation has broken down, then the treatment appropriate to that condition should of course be given. But if this is not the case,—if, for example, there be some fæcal accumulation,—then purgation is clearly called for. Castor oil is probably the best agent for administration by the mouth, but if the accumulation is of any considerable degree, then I think an enema composed of turpentine, castor oil and olive oil, thrown high up the rectum, is better. This should be of a quantity capable of retention, and should be followed in two hours by a large

Purgation.

injection of hot water. In any case of this description a regular and rather free movement of the bowel should be maintained by appropriate means, such as a daily dose of Carlsbad salt or some alkaline water. This will tend to relieve any venous engorgement which may be present, and you may find that such simple remedies are of themselves sufficient to cut short the neuralgia. But if the plethora is very distinct, it may be wise to use more energetic means, such as the application of leeches close to the anus, or wet cupping over the nerve, particularly over the points most tender to pressure.

Effects of acting on bowel.

It may well be that the good effects of emptying the lower bowel thoroughly in cases of sciatica depend, sometimes at least, on that curious inter-relation of the visceral and somatic nerves of the same segment which has been already mentioned in connection with "referred pain." Just as irritation of the skin area corresponding to a particular spinal segment relieves pain in connection with the viscus which stands in relation to that segment, so we may reasonably suppose that conversely irritation of any viscus (in this case the lower bowel) may tend to remove pain in the corresponding somatic segmental area.

It is hardly necessary to say that in all such cases the diet should be carefully considered. It should be ample, but its constituents should be light and easily digested. *Diet.*

The causes of sciatica may, however, not be at all of a mechanical kind. Undoubtedly the condition very often arises from the action of toxines of various sorts. Indeed in some cases of constipation and fæcal accumulation, perhaps even in most of them, the true cause of the sciatica is that variety of auto-intoxication which follows the absorption of poisonous toxines from the intestinal tract. This is so frequent and likely that in all such cases, in addition to purgation, it is well to give some gastric and intestinal antiseptic. I have alluded to this topic already when speaking of trigeminal neuralgia. *Toxines.*

Nor is it needful to refer further or more specifically to such poisons as lead, alcohol, syphilis, gonorrhœa and the like. The line of treatment in cases in which these are acting is sufficiently apparent. Nor need I, in referring to diabetes as a cause of sciatica, do more than mention that in that disease the pain is apt to be present in both sciatic nerves. Gout is so

Gout.

frequent a cause of seiatica that other evidence of its presenee should be diligently sought for in every ease. Even when no elear indications of the presenee of gout ean be deteeted, I think it wise in eases otherwise unexplainable to direet the treatment as if the diagnosis of gout had been established.

Rheumatism.

Those eases of seiatica whieh follow exposure to cold, sitting on damp ground and the like, are probably rheumatie in their origin, and demand speeial treatment from this point of view.

Relief of pain.

When we pass from the causal indications to eonsider the treatment of the pain itself, the first point to notice is the neeessity of rest. In all recent and acute cases that rest should be as absolute as is attainable. The patient should be kept lying in bed, and should not be allowed to leave that posture for any purpose. You will find that he naturally places himself on his side, with the knee on the affected side flexed. This is obviously done to remove strain from the nerve, and in all acute eases this is the posture to be recommended. In ehronic cases, however, it is often well to secure immobility of the limb by applying the long splint. I have seen most excellent results produced, even in obstinate

Rest.

Use of long splint.

cases, by a sojourn of some weeks in such an apparatus.

Heat. The pain is often soothed by external applications. Some people use cold and apply ice-bags over the nerve. But heat is better, and a hot bag of salt is usually very grateful. The more thorough heating which may be procured by means of an electric-light bath I have found very efficacious. But the simplest form of apparatus for applying dry heat is that of Bier. *Bier's method.* According to his method the patient lies with the affected part surrounded by a wooden, asbestos-covered box, the air in which can be raised by a gas jet to any desired temperature. Most admirable results are to be obtained in this way. In some cases also great relief follows the application of the hot pack, hot douches and the like.

Counter-irritation. In subacute and chronic cases much good may sometimes be effected by counter-irritation over the nerve, particularly over the tender points. The simplest plan is to use the faradic brush, and this is often of great service, though in many cases the effect soon passes off. If this fails, blisters should be tried, and sometimes the actual cautery is required.

Acupuncture. There is another plan, viz. acupuncture,

derived, I believe, from the Far East—China and Japan—which I have practised for many years, and which is often wonderfully efficacious. In using this method the patient is placed in a recumbent posture on the face, and sterilised needles, about three inches in length, are passed through the skin in such a position as to transfix the sciatic nerve. The needles I use are of steel, and I usually put in four or five of these, at one sitting, at various points in the course of the nerve. They should be left in position for some minutes. The relief of pain which follows this procedure is often astonishing. One imagines that the beneficial effect is due in part to counter-irritation and possibly also to the piercing of the sheath of the nerve, which may allow of the escape of exudation. But whatever the reason may be, I can assure you from long experience that the method is a very useful one.

Nerve stretching.

The value of nerve stretching in the relief of the pain of sciatica is considerable. Many years ago surgeons used to cut down on the nerve, and, having exposed it, make considerable mechanical traction both on its upper and lower portions. This operation is hardly ever performed now. The nerve can be stretched quite sufficiently

without any surgieal operation. All you need to do is to dorsiflex the foot of the patient at the ankle, to extend the leg at the knee, and then to flex the whole limb at the thigh. As the patient lies on his back with the pelvis fixed, the knee is thus brought near to the sternum. Considerable relief of pain may be brought about in this way.

Electricity may also be employed in allaying the pain of sciatica. I have already mentioned the counter-irritative action of the faradic brush. But more lasting effects may be obtained by means of the galvanic current. What I am in the habit of doing is to apply the negative electrode, which should be of large size, to the sacrum, and the positive pole, also in the form of a large eleetrode, over the point of exit of the nerve and afterwards at any painful points over the course of the nerve which may be discoverable. The current should be put on gradually by means of a rheostat. Its amount should not exceed 5 m.a., and the duration of each application should be from five to ten minutes. Some physicians prefer to use a specially eonstrueted electrode, which is passed into the rectum, the lower bowel being previously filled with hot water. I have no experience of this speeial *Electricity.*

method. High frequeney eurrents are also useful, and I have seen most exeellent results from statie eleetrieity in obstinate eases of seiatiea.

Massage. When the aeute stage has passed away a little mild massage over the nerve may usefully be eommeneed. Later on the massage should become vigorous over all the museles of the limb. Indeed a general massage, and even a regular rest-eure, may have to be adopted in appropriate eases.

Drugs. In regard to treatment by means of drugs there is but little to add to that whieh has already been said with referenee to trigeminal neuralgia. The pain may sometimes be relieved by quinine or by phenaeetin and its allies, just as is the case in other forms of neuralgia. And in seiatica, as in trigeminal neuralgia, one does not use morphia so long as its employment ean possibly be delayed. Other remedies should be tried first, ineluding eodeine, and espeeially dionine. Injeetions of earbolie aeid made in the immediate neighbourhood of the nerve have sometimes been of serviee, and the use of osmie acid, applied in the way deseribed in eonneetion with trigeminal neuralgia, may also be tried in difficult cases.

There are, however, two remedies of which special mention ought to be made in connection with the treatment of sciatica, viz. turpentine and iodide of potassium. Turpentine is particularly useful. It should be given in doses of from 5 to 15 ♏, either in capsules or in the form of an emulsion, made up with yolk of egg. The mode of its action is unknown, but one may be permitted to think that it does good by checking abnormal or excessive fermentative changes in the alimentary tract. *Turpentine.*

Iodide of potassium is also specially useful in sciatica, even in cases which are not of specific origin. It should be given in full doses. *Iodide of potassium.*

In sciatica it is not often necessary or advisable to call in the aid of the surgeon unless the pain be a mere symptom produced by a removable tumour. There are, however, instances in which adhesions have involved the nerve sheath, with resulting obstinate sciatic pain. In such cases the nerve should be exposed by incision and its sheath freed from any adhesion which may have bound it down to neighbouring tissues. *Surgical measures.*

It does not seem necessary to refer more specially to the other forms of neuralgia which

you will encounter from time to time in the course of your work. What has been said regarding trigeminal neuralgia and regarding sciatica in respect of the treatment of these two maladies applies, *mutatis mutandis*, to the other neuralgias.

Lightning pains.

A word may, however, be said regarding a severe form of nerve pain—namely, the lightning pains of locomotor ataxia—though this symptom is not usually classified along with the true neuralgias. These lightning pains are, as you know, very commonly present in cases of that malady. They often assume a conspicuously severe character, and demand careful treatment.

And their treatment.

As a general rule, sooner or later, one has to have recourse to the subcutaneous injection of morphia to remove for the time the agony from which the patient is suffering, but its use should be delayed as long as possible. Locomotor ataxia is, as you are aware, a very chronic malady, and its subjects are unusually prone to

Use of morphia.

become morphinists. Therefore, you should be careful to keep the administration of morphia in your own hands, and to stop its use when, as is usually the case from time to time, an interval of longer or shorter duration occurs in which the patient is free from his lightning pains.

But before having recourse to morphia, you should always try the effect of lighter remedies. Counter-irritation by means of liniments or the faradic brush, massage, or wet packs may in slight cases be of service. As a rule, however, you will find these of little or no use. Of more effect is the administration of phenacetin or of antipyrine, pyramidon, aspirin, salicylate of soda. Whichever of these remedies you select should be given in full doses, the effect being carefully watched. If these and the other drugs of this class fail, then you should try the morphia derivatives. Codeine sometimes answers well, and in dionine we have a very reliable substitute for morphia. It may be given by the mouth in doses of one-third to two-thirds of a grain. But in too many cases you will find that your hand is forced, and that you are obliged to still the pain by means of morphia.

And other remedies.

The condition which we know generically as "headache" is one of the most frequent symptoms met with in practice. It may arise from many and varying causes, and the treatment which should be adopted ought in each case to be that of the malady of which the cephalalgia is a

Headache.

symptom. To discuss the subject generally is clearly outside the scope of these lectures; it would indeed involve a scrutiny of many of the problems of general medicine, and would lead us far from those topics which are usually included under the term of nervous disease.

Migraine. There is, however, one variety of headache, namely migraine or hemicrania, which is certainly pertinent to our present subject. Migraine is indeed very distinctly a neuropathic disorder. It is essentially hereditary, being transmitted through certain families in a notable degree. In this respect it resembles epilepsy and the other *Heredity.* diseases met with in neuropathic families; indeed its associations with epilepsy are singularly close, for the epileptic convulsion is occasionally replaced by an attack of migraine.

The hereditary condition which renders an explosion of migraine easy in certain individuals, so overshadows the clinical picture that the *Proximate causes.* proximate or exciting cause is for the most part not prominent nor easy to recognise. That such attacks result from the strain of intense mental work is certainly true in some cases. In others, digestive disturbances and the resulting auto-intoxication act as the starting-point. Errors in

connection with the visual apparatus as well as the metabolic changes of gout, may also be mentioned in this connection.

Symptoms. The disease consists of periodic attacks of headache of greater or less severity, the pain being usually at first limited to one side of the head and attaining its greatest severity in the frontal region above the orbit. It is aggravated by movement, by light, and by sound, and is accompanied with mental oppression. The attack lasts a varying time, its duration seldom, however, exceeding twelve or fifteen hours. It is usually accompanied with gastric symptoms, and often with vomiting.

Along with this headache there occur other nervous symptoms. Several of these appear to be due to some disturbance of the functions of the sympathetic system. *Sympathetic symptoms.* In most cases these signs are those of irritation: pallor of the face, contraction of the temporal artery, dilatation of the pupil, slight retraction of the globe of the eye, and perhaps some salivation. Sometimes, however, the symptoms are rather those which we associate with paralysis of the sympathetic, namely, flushing of the skin of the face, dilatation of the arteries, contraction of the pupil and slight exophthalmos.

Visual symptoms. There may also be subjective visual symptoms, such as scotoma, particularly that zig-zag, scintillating form which has been compared to the outline of a fortification. Various other symptoms may occur in cases of migraine though they are not invariably present. Among these may be mentioned giddiness, paræsthesiæ of divers kinds, and temporary and very transitory aphasia.

In those subject to migraine the attacks come on at varying intervals. Days, weeks, or even months, may intervene, and as the patient becomes older there is a tendency towards recovery and the attacks become less and less severe.

Treatment From what has been said it must be clear that during a severe attack the patient should lie down in a darkened and quiet room, so as to avoid as much as possible those stimuli—movement, light and sound—which tend to aggravate his symptoms. You hardly need to tell him to *General measures.* adopt this course. He does it naturally and as the result of past experience. The pain may sometimes be relieved by purgation and the *Purgation.* attack thus cut short. This, however, so far as my experience goes, will only happen if the purge

be taken at once, whenever the symptoms begin to show themselves. If time is allowed for the attack to develop, this method of treatment is not of much avail. The severity of the pain may usually be mitigated by the administration of phenacetin, antipyrine, pyramidon, or other member of this group of drugs. On the whole I have had best results from phenacetin in rather full doses—15 grains or even more—which may be repeated once or twice, and with which I am in the habit of combining caffeine. In cases of marked severity and when the headache is overpowering, such remedies, even when given in large doses, may fail, and you may have to resort, however reluctantly, to the hypodermic use of morphia.

Pain relievers.

The pallor of the face and the vaso-constriction of which it is the result suggest the use of such remedies as cause vaso-dilatation, and sometimes the administration of nitrite of amyl, nitroglycerine or erythrol tetranitrate may afford relief.

Treatment in the intervals.

Apart from such measures as have been mentioned, which are fitted for use during the attack of migraine, we have to consider the general treatment of the underlying condition. The

lines to be adopted are very much those to which I shall refer later when speaking of the management of neurasthenia. The patient may do much towards warding off these attacks by a sensible regulation of his mode of life. He should avoid the use of alcohol and of tobacco. His diet should be a simple one and should not include much red meat. He should live an active life, take plenty of exercise, avoid late hours and limit the intensity and the amplitude of his mental work. Attention to the state of the bowels is of importance, and an occasional laxative is usually needed. If there are evidences of the presence of gout, the treatment ought to be directed in accordance with this indication. In some cases of migraine a course of bromide medication is advisable.

LECTURE VIII

ON THE TREATMENT OF THE DISORDERS OF CO-ORDINATION AND EQUILIBRATION

ATAXIA AND VERTIGO

Disorders of co-ordination—Recent advances as regards treatment—Underlying physiological arrangements—Definition of co-ordination—Its mechanism—Centripetal and centrifugal paths—Centres for co-ordination—Definition of muscular sense—Its component parts—Its disorders—Ataxia—Vicarious action of other neurones—Curative effects of attention and practice—Treatment of tabes, its practical application—Details of Frenkel's method—General management of tabetic cases and the treatment of special symptoms.

Vertigo, etiology and method of its production—Ménière's disease, its causes and treatment.

Disorders of co-ordination and equilibration.

In the whole wide field of neurology there is, to my mind, no subject which equals in interest that involved in the consideration of those disorders which affect the mechanism whereby co-ordinated actions are performed and the erect position of the body is maintained. Intimately related to these—indeed in some measure overlapping them—stand the maladies which produce

objectively a very marked loss of equilibration, and subjectively that symptom known as vertigo.

These two sets of diseases are so mixed up, as you will presently see, that it is hardly possible to consider one without some reference to the other. We shall, however, commence by studying those disorders of which the main symptom is loss of co-ordination. The classical example of this group of diseases is tabes or locomotor ataxia, the main lesion of which is one involving the centripetal paths conveying muscular-sense impressions. These neurones pass, as you know, from the periphere in the mixed nerves, enter the cord in the posterior root and make their way upwards, in the manner which will be indicated presently, to the medulla, to the cerebellum, to the optic thalamus, and to the cortex. The tabetic lesion is mainly one of the peripheral neurones of muscular sense, to some extent in the nerves, but more distinctly in the prolongations of these neurones up the spinal cord. The same neurones may, however, be attacked by other kinds of toxine. The auto-intoxication from faulty metabolism seen in pernicious anæmia, and in diabetes, sometimes attacks

Locomotor ataxia.

Tabetic lesion.

Toxine lesions of various kinds.

them and leads to symptoms resembling in many respects those of tabes. Ergotine and sundry other poisons may act in a similar way. In some diseases the more peripheral part of these neurones may be peculiarly involved. In multiple neuritis, for example, the toxine may act almost exclusively on the fibres conveying muscular sense. I have also seen a similar selective action in cases of typhoid fever and of influenza. In the year 1893 Dejerine and Sottas described a curious family disease, interstitial hypertrophic neuritis, in which the neurones of muscular sense specially suffer; and there is another rare disease, also described by Dejerine, "nervo-tabes périphérique," of which the same may be said.

Interstitial hypertrophic neuritis.

These fibres may, however, be affected higher in their course, and you may have an ataxia proceeding from a medullary, from a cerebellar, or from a cerebral lesion. These may, however, be combined in various ways. In Friedreich's ataxia, for example, there is in all likelihood a developmental defect leading to loss of vitality and early decay of the muscular sense neurones in the cerebellum and cord. A very high degree of ataxia is produced by lesions of the

Friedreich's ataxia.

Cerebellum lesions.

cerebellum itself, especially of the middle lobe; and it seems probable that alcohol, in producing ataxic reeling, does so by acting on the cerebellum. Very profound changes of this kind follow lesions of the semicircular canals and of the vestibular portion of the eighth nerve. These, however, will be studied when we come to consider the varieties of vertigo.

Ménière's disease.

Dealing in the first place with the various forms of ataxia, we shall find that, so far as the interference with co-ordinated movement is concerned, the method of treatment which ought to be adopted is very similar in the case of each of these maladies. This method will now be considered. Thereafter we may discuss the various indications for treatment, other than that directed against the ataxia, which these maladies present. We shall, finally, consider the measures to be adopted in the treatment of the various forms of vertigo.

Results of treatment in cases of tabes.

In no department of neurology has greater progress been made of late years in the direction of therapeutic measures than in that which concerns the group of lesions involving the apparatus of co-ordination and orientation. While, a few

years ago, we stood nearly helpless before a case of advancing locomotor ataxia, knowing that ere long the patient would almost certainly become bedridden, we now stand in an altogether different position. We know that, for many years at all events, we shall be able to ensure that he can move about, that the advancing ataxia will be held in check, and that to a considerable extent the power of co-ordinated movement will be regained. The advantage of this to the patient consists not merely, or indeed mainly, in the comfort and satisfaction it gives him, but more importantly in that he can have the advantage of moving about in the open air, and that the danger following the formation of bed-sores is avoided, or at least postponed for an indefinite period.

We owe this great advance in therapeutics to the labours of a Swiss physician, Dr. H. S. Frenkel, who with skill and perseverance has developed the method of what may be called compensatory therapeutic exercises. By this system of treatment, which consists in carefully designed and regulated exercises, the ataxia is to a large extent reduced and the power of co-ordinated movement correspondingly re-established. The

Frenkel's method.

regaining of this co-ordinating function, which, when observed clinically, is a very striking phenomenon, is due, not to the re-education of paths which the degeneration has affected, but to the substitution of neurones capable of acting vicariously, and probably to the education and opening up of new paths through which the centripetal impressions necessary for co-ordinated movement may pass to the higher centres. The results depend also to a considerable and important extent on that increased acuteness in the regulating centres which practice brings with it.

On what it depends.

To understand this subject and still more to be able to apply the method accurately and suitably in particular cases, it is necessary to consider with some degree of minuteness the nervous mechanism by which muscular movements are co-ordinated.

Definition of co-ordination.

By a co-ordinated movement is meant one which fully and exactly fulfils its purpose, and yet does so without any undue expenditure of nervous or muscular energy. To arrive at this precision of result it is necessary that certain muscles should be set in action in a certain order and with a certain degree of force, which degree may, in the case of each muscle, have to be

modified—diminished or increased—during the course of the action.

The matter is therefore very complicated, for no action is performed by means of one muscle alone. There are always others which come into play either by contraction or by relaxation. Take for example the action of grasping with the hand, which Duchenne so carefully studied. In a firm grasp the fingers are flexed in the palm, but at the same time the hand is extended at the wrist. This synergic movement has for its purpose the increasing of the distance between the points of origin and insertion of the flexors, thus enabling these muscles to act more effectively. We have here, then, an example of a movement so co-ordinated as to make the grasp of the hand more useful to the organism. Another example of co-ordination is to found in the relaxation of antagonistic muscles when certain actions are performed. Sherrington,[1] in the course of his observations on the effects of

Synergic action.

Relaxation of antagonists.

[1] Sherrington, "Note on the knee-jerk and the correlation of action of antagonistic muscles," *Proceedings of the Royal Society of London* (Feb. 9, 1893).

"Further experimental note on the correlation of action of antagonistic muscles," *Proceedings of the Royal Society of London* (May 4, 1893).

"On reciprocal innervation of antagonistic muscles," *Proceedings of the Royal Society of London* (Jan. 21, 1897).

stimulating the cerebral cortex in the monkey, noticed that when any particular point of the Rolandic area was stimulated there was not only contraction of the muscles concerned in performing the special movement which that part of the cortex subserved, but also a simultaneous and marked relaxation of the antagonists. The fact that along with the contraction of the agonists there is a simultaneous relaxation of the antagonists has since been confirmed by many observers.[1] You may indeed satisfy yourselves without difficulty that this relaxation does occur. If you bend the foot dorsally at the ankle and then, against a firm resistance, try to flex, you will feel that the muscles of the calf contract strongly, while, if your finger is laid upon the extensors, you will be able to satisfy yourselves that their fibres simultaneously relax. I do not suppose that in every movement there is this relaxation of the antagonists. In some cases it is more useful for the due carrying out of certain movements (in drawing, for example)

Correlation of agonists and antagonists.

Example.

[1] Hering and Sherrington, "Ueber Hemmung der Contraction willkürlicher Muskeln bei electrischer Reizung der Grosshirnrinde," Pflüger's *Arch.*, Bd. 68 (1897), S. 222.

Hering, "Beitrag zur Frage der gleichzeitigen Thätigkeit antagonistisch wirkender Muskeln," *Zeit. f. Heilkunde*, Bd. xvi. (1895), S. 129.

that the limb should be held rigid, and in such cases probably the antagonists contract to some extent.

Complexity of mechanism. Into this question, however, we do not need to enter further here. What has been said is intended to show the great complexity of the mechanism required for the performance of even apparently simple actions. It is to be specially noted that in the course of such synergic movements it often happens that muscles are simultaneously engaged, the spinal centres of which are not in immediate proximity—for example, the flexors and extensors in the act of grasping.

For the performance of these comparatively simple examples of co-ordinated action which have been given, and still more for the many more complicated and finer actions of daily life, a very delicate nervous mechanism is obviously required. *Centres for co-ordination.* The centres concerned in the co-ordination of muscular movements lie mainly in three places; in the spinal cord, in the cerebellum and in various nuclei in its vicinity, and finally in the cerebral cortex.

Their modification. The mode in which these centres act is probably twofold. For the finer actions to

which education has brought us, such as speaking, writing, drawing, the use of fine tools, piano-playing, and the like, there seems no doubt that the necessary motor memories are stored up in the cortex, *i.e.* memories of how, in what number, order and graduation of strength, the necessary muscles are to be employed in performing an action which has been very frequently repeated. But while this is no doubt correct for such actions as those mentioned, for which the cortex has been highly educated, it does not complete the mechanism even for them, and to the numberless other actions of daily life, including walking and equilibration, it does not apply. For all centres concerned in the carrying out of co-ordinated actions stand under the control of centripetal connections of a complex kind, and they cannot innervate the muscles correctly without the guidance of the impressions these centripetal neurones bring.

Motor memories.

Centripetal connections.

The motor cells, whether in the cortex or in the anterior horns of the cord, can, by their stimuli, cause the muscles to contract, but no co-ordinated action can be performed unless those centripetal neurones which have to do with orientation, equilibration and muscular

Necessity of centripetal impulses

sense are in function, in order to direct and control those motor centres.

So that, so far as the phenomena of co-ordination are concerned, we have to look not so much to the motor centres as to those centripetal neurones and to certain centres to which they stand in special relation.

Nervous mechanism used in executing a voluntary movement.

To put the matter simply, we may consider what happens when we will to make a movement of the hand. The stimulus passes from those centres where the Will resides to the cortical motor cells governing arm movements. From these the stimulus goes through the pyramidal tract and the anterior horn of the cord to the various muscles to be employed. But it is clear that these muscles cannot be accurately used or controlled unless the centres are informed in some way as to what, at the moment, is the position of the limb, to what degree the joints are flexed, to what extent the muscles are contracted or relaxed. In the case of the arm this information comes in two ways—by the nerves conveying muscular sense, and by the optic nerves. Of these the former are the more essential and important, for a healthy man can and usually does carry out arm movements

Centripetal impressions.

without using his eyes. It is only when the neurones conveying muscular sense have undergone degeneration that he requires to watch the movements of his hand and so direct them. An exception to this statement is to be found in the case of very fine movements, such as writing, where accuracy requires that both muscular sense and visual neurones must be employed in order that the centres may perfectly co-ordinate the action.

Various centripetal paths used in co-ordination.

The chief ways in which the regulating centres gain such information as shall enable co-ordinated movements to be carried out, are as follows:—

1. The optic nerve.
2. The vestibular branch of the eighth nerve bringing impressions from the semicircular canals.[1]
3. The neurones conveying "muscular sense" impressions from the periphere.

Varieties of information conveyed.

Of these, the optic nerve gives impressions as to the direction, degree and rapidity of a move-

[1] The auditory branch of the eighth nerve ought to be included in this list, but, to avoid unnecessary complication, this has not been done. It is used to some slight extent in orientation, but in so far as co-ordination is concerned, these acoustic neurones are chiefly employed in connection with the movements of speech and with those of a musician in playing on an instrument.

ment. The vestibular neurones give information as to the movements and position of the head, and, in sitting, standing and walking, of the body generally, such information being derived from the end-organs in the semicircular canals. The neurones conveying "muscular sense" impressions give information as to the position of the limbs, the degree of flexion of the joints, the tension of muscles. For the acts of standing and walking the healthy man does not require to use optical impressions; the vestibular and muscular sense impressions are sufficient, and it is only when one of these is impaired that the help of the eyes is required.

Muscular sense.

The term "muscular sense," which has been used since the brilliant generalisation of Charles Bell,[1] is one including many different centripetal impressions, which, taken together, are sufficient to inform the centres as to the varying position of the limbs.

Tactual impressions.

Important information reaches the centres from the skin, chiefly tactual impressions, arising from varying degrees of stretching and relaxation of the skin over joints, and more particularly

[1] Charles Bell, "On the nervous circle which connects the voluntary muscles with the brain," *Transactions of the Royal Society of London* (1826), p. 163.

when the end-organs are stimulated in any act involving contact or pressure.

These cutaneous impressions do not, strictly speaking, form part of that group of sensations which we know as muscular sense. In cases in which that special sense is in default and at the same time tactual sense is preserved, these skin impressions are fitted to act vicariously in aid of muscular sense. In fact, in strict accuracy, the paths of tactual impressions might have been specifically mentioned in the list, just given, of centripetal neurones used in the process of co-ordinating muscular movement. To avoid needless complication this has not been done. Indeed, so far as clinical work is concerned, the question hardly arises, for the cases are few in which muscular sense fails while tactual impressions are preserved.

True muscular sense impressions.

But the true muscular sense impressions are much more important and essential. These are derived from the surfaces of the joints—indicating the direction, rapidity and degree of excursion of any movement; from the capsules of joints; from ligaments and tendons; and, most importantly, from the muscles themselves. These impressions, taken together, give sufficient

Their points of origin.

information to the centres to enable co-ordinated movements to be carried out. In great measure these impressions do not rise to consciousness; indeed it is obviously not necessary or even desirable that they should do so. It is clearly better that movements should be as automatic in their regulation as possible. But to some extent they do rise to consciousness. Skin sensibility of course does so, and, speaking generally, a healthy man ought to be able to appreciate and define the position of a limb, with no other aid than muscular sense impressions. To some extent those impressions can be analysed, for with care we may elicit and even approximately measure the sensation which comes from joint surfaces. Goldscheider[1] has indeed established the threshold of sensation for angular movement in connection with each joint. Duchenne studied the sensation which arises when a muscle is made to contract by faradism, and Frenkel and Foerster[2] have determined the threshold of that sensation. Then there are the feelings of which we are conscious when muscles are passively

Chiefly unconscious.

Perception of angular movement.

Perception of muscular contraction.

[1] Goldscheider, *Gesammelte Abhandlungen*, Bd. ii. S. 290.

[2] Frenkel and Foerster, "Untersuchungen über die Störungen der Sensibilität bei der Tabes dorsalis," *Arch. f. Psychiatrie*, Bd. 33 (1900), S. 108.

stretched, and, further, the sensation of muscular fatigue. These all are component parts of muscular sense. Egger's observations regarding the sensibility of the bones of the limb as tested with a tuning-fork are of great interest, because the sensation produced by the stimulus passes through the joint surfaces. I do not think, however, that it can be regarded as a test of muscular sense, for though, when muscular sense is lost, bone sensibility is usually correspondingly lost, yet I have not found this to be invariably the case.

Fatigue.

Bone vibration.

It has been said that there are mainly three centres in which movements are co-ordinated—the spinal cord, the cerebellum with certain nuclei in its vicinity, and the cerebral cortex. As regards the first of these—the spinal cord—it is of course certain that in the lower animals co-ordination is performed there. A decapitated frog, for example, jumps and swims with apparently perfect co-ordination. How far this may be true as regards man is doubtful, but in all likelihood certain automatic actions which require to be very rapidly performed are co-ordinated in the cord. For example, if the foot slips on a stone the first attempt to correct its position by

Centres governing co-ordinated movement.

Spinal cord.

muscular contraction comes in all likelihood from the centres in the cord.

Cerebellum.

But the consideration of the process of co-ordination in the cerebellum is much more important. The various avenues by which information passes as to the position of the body and its various parts, centre themselves in the cerebellum and chiefly in its middle lobe.

Course of fibres conveying muscular sense.

The fibres conveying impressions of muscular sense enter the cord in the posterior nerve roots and pass upwards through the posterior columns to the nuclei of Goll and of Burdach. Similar impressions also pass in the fibres of the direct cerebellar tract and in those of Gowers' tract. From the medulla the muscular sense impressions gain the middle lobe of the cerebellum through the restiform body. It is to be noted that muscular sense impressions from the muscles which move the eyeballs also reach the cerebellum, and the knowledge so attained as to the exact position of the eyeballs, taken in conjunction with retinal impressions as to the corresponding position of external objects, gives very important aid in the orientation of the body.

Vestibular impressions.

To the cerebellum come also the very important impressions conveyed from the semi-

circular canals by the vestibular branch of the eighth nerve. These are, of course, very essential to equilibration.

Thus from three sources—the muscular sense, the visual sense, the impressions from the semicircular canals—the cerebellum receives information such as enables it to maintain equilibrium and co-ordinate muscular action.

Centrifugal fibres of co-ordination.

This is not the place to attempt to give exact anatomical details, nor indeed is our knowledge as yet sufficient for this purpose. It may, however, be said generally that the centrifugal stimuli subserving co-ordinated movement pass from the middle lobe of the cerebellum, and probably also from the red nucleus, to the nuclei of Deiters and of Bechterew, and thence down the cord to the anterior horns. These fibres probably go by way of the vestibulo-spinal and rubro-spinal tracts. Thus the movements necessary for equilibration are carried out and the governing of co-ordinated action performed.

Example of cerebellar and cerebral control.

Let us consider, by way of example, how these various mechanisms act in a particular case. Let us suppose that for some accidental reason the body, while standing, sways so much in some direction that the balance tends to become

lost. This fact is at once communicated by the vestibular neurones. The cerebellum responds by sending down the cerebello-spinal tracts such stimuli as shall cause those muscles to contract which are necessary to restore the balance of the body. For this purpose, however, the vestibular information is not sufficient. That information goes only so far as to indicate that the balance is being lost in a particular direction. From the neurones of muscular sense, however, the cerebellum learns what joints and what muscles are at fault, and is thus in a position to apply the necessary correction. The cerebellar action is thus a purely reflex and automatic one. For moderate degrees of loss of balance it suffices, but if the amount of swaying is considerable then the conscious action of the cerebrum comes in. This cerebral action is partly direct, the necessary muscles being caused to contract by stimuli passing from the motor cortex down the pyramidal tracts. But in addition to this, the cerebrum probably stimulates the cerebellum to increased action through the cortico-cerebellar tracts.

Vestibular impressions.

Muscular sense impressions.

Automatic cerebellar action.

Reinforcement by cerebrum.

Turning our attention now to the patho-

Results of pathological changes.

logical side of the question, let us consider what will be the result of lesions interfering with these various centripetal paths and with the centres which govern co-ordinated movement. These results differ, of course, according to the situation of the lesion. In Ménière's disease, for example, the labyrinthine disturbance produces profound vertigo, the impressions coming through the vestibular neurones being, as it were, contradictory and no longer indicating with any exactness the position of the head. A similar vertigo with characteristic reeling follows lesions of the cerebellar centres, whether these lesions be of an organic nature or whether they result from the temporary action of some poison, such as alcohol.

Ménière's disease.

Cerebellar lesions.

Lesions of muscular sense paths.

As regards treatment, however, what chiefly concerns us is the consideration of the lesions affecting the neurones conveying centripetal impressions of muscular sense. At whatsoever point in the long course of these neurones the lesion occurs, the result is essentially the same. Whether the peripheral fibres be affected, or those in the cord, or those in the medulla or still higher in their course, the predominant resulting symptom is ataxia. And by ataxia we mean a

Ataxia—definition of the term.

condition of matters in which, when a movement is attempted, the necessary muscles are incorrectly innervated, the contraction brought about being either too strong or too weak. Or it may be that the various muscles do not pass into contraction in the exact order which is necessary. And, finally, the stimulus may pass to the wrong muscles. In any case the movement ceases to be a co-ordinated one.

Tabetic symptoms.

Ataxia finds its classical example in tabes, and it is in connection with this disease that it has been most frequently studied. When the tabetic symptoms are well developed, the movements of the patient are inexact and ataxic to a high degree. This results from the failure of muscular sense impressions, conscious and unconscious, due to the tabetic lesion. What strikes the observer at first is that each voluntary movement is carried out with abnormal quickness and with quite unnecessary force. In walking, for example, the excursions of the limbs are needlessly great, and the patient brings his heel down to the ground with unnecessary force and with the stamp which is so characteristic a symptom. In health, when a voluntary movement is carried out, the centres receive

Characteristic movements.

Gait.

certain sensations (chiefly probably unconscious) which indicate that the proper degree of flexion of the limb has occurred, and that the requisite amount of muscular contraction has been carried out. These, which are muscular sense impressions, are greatly weakened in a tabetic man. But as his centres have been accustomed during years preceding the onset of disease to receive normally strong impressions from muscular movements, these centres are, as it were, now deceived and misled by the faintness of the muscular sense impressions which are reaching them. Hence they automatically continue to stimulate the muscles to increased and, in this case, unnecessary effort.

Weakening of muscular sense impressions.

The loss of intensity of muscular sense impressions in locomotor ataxia also acts in another way so as to produce the exaggerated as well as the defective and erring voluntary movements which one sees in that disease. When the movement is initiated, the centres do not know with accuracy the momentary position of the limb. For this reason they cannot appreciate the distance through which the limb has to be moved in order to attain the object of the movement. Hence come exaggerated and

Want of knowledge of position.

defective excursions. Thus, in order that a tabetic may regain the power of co-ordinated movement he has much to learn and also much to unlearn. He must by careful practice and attention teach his centres, as it were, that the feeble muscular sense impressions which are now reaching them are expressive of muscular movements of a degree and intensity from which formerly very much stronger centripetal impressions would have come.

Vicarious action.

The success which attends the training of a case of locomotor ataxia depends mainly on the fact that the co-ordination of movement, resting as it does on centripetal impressions, does not rely on one set of impressions alone. Vestibular, ocular, muscular sense-impressions are made use of, and when one fails, the others come to the aid of the organism. And even with regard to muscular sense itself, these impressions, as I have already said, pass to the regulating centres by various paths, so that if one path is blocked by disease, there remain others—possibly not so direct or so quickly serviceable—but still capable of being made use of so as to compensate for that which has been destroyed. It is also possible that under such circumstances new

Other paths available.

New paths.

paths may be opened up, and neurones not usually employed for the passage of muscular sense-impressions may be utilised for that purpose.

Examples of vicarious action in general medicine.

This vicarious action pervades the whole field of medicine and becomes apparent in all clinical work, whatever may be the system of the body which is affected. When, for example, excretion by way of the kidneys is difficult, elimination may be effected vicariously by skin or by intestine. In cases in which the functions of the stomach are in default, the duodenum may and does take on part of the work. When one set of glands has been destroyed another set of correlated glands enlarge in order to act vicariously. I might go on almost indefinitely in adducing examples of this vicarious action. Its highest development is, however, to be found in connection with the neurones of which we are now speaking.

Automatic vicarious action.

This substitution of one set of neurones for another set which has been destroyed or at any rate weakened in function, occurs to some extent automatically. A patient suffering from early tabes becomes ataxic (in the strict sense) before he knows it. He can still walk with apparently

perfect ease and fair accuracy, but he does this by unconsciously making use of his eyes, and it is only when he steps into a dark room where the visual neurones can no longer help him that he becomes aware that neither standing nor walking is possible. If he belongs to that portion of humanity which washes its face, he may very likely learn of the onset of ataxia by noticing that when he holds his eyes closed he tends to fall toward the basin.

Limitations of the action of nature.

But the compensation which Nature provides does not go very far. For soon the tabetic, if no treatment is attempted, becomes profoundly ataxic and ultimately bedridden. A special spur to care and attention is required to aid the failing sensations. This spur is sometimes supplied naturally. Think for a moment to what acuteness the other senses can be brought in the case of a blind man; how, in spite of his infirmity, he can still guide himself; how acute his hearing becomes, how refined his sense of touch. And so, if a tabetic suffers from early optic atrophy, a similar spur urges him to increasing effort in the way of attentive practice, and it is long before he becomes profoundly ataxic.

Use of attention.

By careful attention all the faculties become more acute, and in this lies part of the secret of the success of Frenkel's method of treatment. In this way all nervous mechanisms become more refined in their action, sensations are more acutely perceived, and complicated reflexes are more sharply and accurately carried out. You are all aware that pain is more acutely felt if the mind is allowed to dwell on it, and that it becomes more bearable when the attention of the patient is diverted. Indeed any sensation, if you look for and attentively expect it, is more easily perceived than if it comes unexpectedly. How acute the hearing becomes when one is eagerly listening for the footstep of a friend. And in a similar way, but in the other direction, diminution of the power of attention lessens the acuteness of perception. I have already[1] told you how, in the case of children, the exhaustion of school work blunts the tactual sensibility to a very considerable and measurable degree.

Refinement of reaction it produces.

Further effects of attention.

Close attention, then, stimulates the receptive centres, and probably this does not limit itself to those which consciously perceive, but in all likeli-

[1] See page 29.

hood extends to perceptions which lie beneath the threshold of consciousness. Nor does attention sharpen perception only; it also refines and gives acuteness to the act of motor execution.

Importance of attention in tabetic patients.

Just as, in a healthy man, careful attention sharpens perception and refines action, so in a proportionate degree does a similar attention enable a pathologically weakened sensation to be perceived. In this way the faint muscular sense impressions which still pass up the cord of a patient suffering from locomotor ataxia can be raised, so far as perception is concerned, to a degree sufficient to enable co-ordination to take place.

Curative action of attention.

The curative action of attention, then, is of very great importance, and it is a fortunate circumstance that the refinement of perception and of execution which regular attention and practice bring, is not fugitive, but on the contrary persists, and is to such an extent retained that after some little time the co-ordinated action can be performed automatically and almost without attention. You will see what I mean if you contrast the act of writing in the case of a highly-educated and of an ill-educated man. The former, though no doubt he uses his eyes,

Refinement of movement which practice gives.

can write without strong effort, and without any very close attention to the mechanical part of the act. If it be necessary, he can even write fairly well in the dark. An ill-educated man, on the contrary, writes only with close attention, slowly and laboriously. Indeed so great is the expenditure of energy in his case that it irradiates on the cortex, and you may see his tongue protrude and move in sympathy, as it were, with his painful efforts as a scribe. *Its persistence.* Just as the eye of a sailor becomes more acute in its visual perception from constant practice and attention, and remains acute, so is it in the case of the impressions of muscular sense and the execution of co-ordinated movement. These become more exact and sharp as the result of close attention and practice, and the increased refinement of this mechanism, so obtained, persists.

Treatment of tabes.

Thus in the treatment of ataxia three elements come into play which more or less blend the one with the other.

Chief elements.

1. Compensation or the use of other centripetal paths to replace those which have become affected by disease.
2. Attention—leading to greater acuteness in the perceptive and executive centres.

3. Regular and attentive practice of those particular movements which have become defective, until they can be performed without close attention.

Thus, when we come to consider how the method is to be practically employed, it becomes clear that in any individual case one must study the movements of the patient so as to determine which particular movements are defective. Take, for example, the case of a tabetic patient who can still walk, though with difficulty. Are the movements of flexion at the ankle-joint defective, or are those of extension imperfect? In the same way you should consider the movements at the knee and at the hip. Having then determined what is defective, you should take each movement separately. The patient is told what is wrong, is shown how the movement ought to be executed, and, watching the foot carefully, he is made to practise the movement until he can do it exactly with careful attention and with the compensatory aid of his visual neurones. As he continues his practice, however, he will find that his centres have now become so re-educated that he can perform the movement correctly without very careful attention and even without visual aid. *Practical application.*

He proceeds in this way to correct all the defective movements which he has exhibited in the act of walking, and thus regains the power he has lost, at any rate to a very considerable extent.

Frenkel's method of treatment.

When we come to consider practically what lines our treatment should take, in regard to this compensatory training, in a case of locomotor ataxia, it is obvious that, in its details, this must depend on the severity of the case and on the degree of ataxia which is present. But, speaking generally, it may be said that the aim is to educate with particular care those co-ordinated actions which are of special service in the daily life of the patient. As regards the lower extremities, these movements are chiefly comprised in the acts of standing, of walking, of rising from the sitting posture, of sitting down, and of mounting the steps of a stair. If the arms are also ataxic, then the acts to be studied with attention and carefully practised are those of grasping with the fingers, the movements of the arms used in the act of taking food, in putting on and taking off clothing, and those of the fingers and hand in writing. The exact

Its general aim.

ways in which these various muscular movements should be practised are carefully described by Frenkel,[1] to whose work I must refer you for the many details necessary for proper and complete carrying out of his method. Very important and useful information on these points will also be found in Foerster's work.[2]

Practice in the horizontal position.

As has been already said, the procedure to be adopted in these cases must vary according to the degree of the ataxia present. If the patient is unable to stand, then the first exercises must be made while he lies in bed. He should be taught to practise with care and attention the various movements at ankle, knee and hip. These should be performed slowly and with careful attempts at precision. He should, further, be taught to hold the limb in particular positions, first with the aid of the visual neurones, then without, in the manner above described. From these the patient passes on to exercises which may develop the power of standing, of walking, etc.

[1] H. S. Frenkel, *Die Behandlung der tabischen Ataxie, mit Hilfe der Uebung*, Leipzig, 1900. A translation of this work into English was published in 1902 by Rebman, London.

[2] O. Foerster, *Die Physiologie und Pathologie der Co-ordination*, Jena, Fischer, 1902. I have found this thoughtful work of great value in dealing with the whole subject of the disorders of co-ordination, and I gladly acknowledge my obligations to its author.

Risk of over-fatigue.

But you have always to bear in mind that in regard to all these exercises there is considerable risk of overtiring the patient. You will remember that one of the signs of interference with the paths of muscular-sense impressions is that the sensation of muscular fatigue is much blunted or even may not be perceived at all. You cannot, therefore, trust to the feelings of the patient as giving due indication of the approach of over-fatigue. These exercises, particularly if they are performed with that strained attention which is necessary to ensure success, are very exhausting. Great care is therefore required in adjusting their duration. The effect of such strain, though it may not cause perceptible fatigue, will show itself on the circulation, and any distinct rise in the rate of the pulse will inform you that the exercise has lasted long enough.

Frenkel's apparatus.

For practising standing, and particularly walking and certain special movements, Frenkel has devised a number of ingenious forms of apparatus. These are described in his book, already referred to. Others will be found in a work by Goldscheider.[1] These are, however, for

[1] Goldscheider, *Anleitung zur Uebungsbehandlung der Ataxie*. Leipzig (1899).

the most part of a kind unfitted for use outside a hospital or special establishment. It is therefore fortunate that quite as good results may be obtained by simple means. Using chalk, it is easy to mark on a wooden floor the lines along which the patient is to walk, the points at which he is to place his feet, and so on. A little ingenuity will enable the physician to obtain his object by such simple methods.

Exercises for hand and arm.

As to the movements of the hand and arm, appropriate exercises may readily be devised. These should have a direct bearing on those movements which are of most importance and of most frequent performance in daily life. You may begin by instructing the patient to practise slow movements of flexion and extension at the wrist and at the various joints of the fingers, and particularly the movements involved in opposing the thumb to the fingers. He should also practise holding the hand and fingers at rest in various positions. These movements should be made with close attention and at first with the aid of the visual neurones. Later, they should be practised with the eyes closed. The patient should then attempt to grasp such wooden discs as are used in the game of draughts. These he

should move about and arrange in patterns, and he should practise piling the one on the other in rouleaux. The hand and arm movements involved in carrying a spoon to the mouth should next be studied. After these, attention may be devoted to drawing straight lines, and, finally, to writing.

General treatment of tabes.

Turning now to the general management of locomotor ataxia, we may group together the various indications for treatment and the measures to be employed.

Is the patient to be informed of the nature of his disease?

The first practical point is one which arises when we are dealing with a very early case of tabes, in the pre-ataxic stage. Are we to inform the patient of the exact nature of his malady? The public, by its perusal of certain realistic novels and in other ways, has unfortunately become aware of the serious prognosis attachable to locomotor ataxia. Consequently it is well to pause before you pronounce a sentence which the patient will consider to be one of death. You will remember that in many cases an accurate diagnosis may be made years before any ataxia manifests itself. I can well recall a case in which gastric crises commenced

eleven years before any sign of ataxia appeared, and yet the conjunction of this symptom with the Argyll Robertson pupil was sufficient to lead to a diagnosis of tabes. It would obviously be wrong to load the patient with anxiety during these years, though it might be wise to mention your suspicions to some judicious relative.

Need of caution. In any case you should hold your hand for some months, for it occasionally happens that signs exceedingly like those of early tabes may arise from other causes. I am reminded of a case which, I think, illustrates this point very well. *Illustrative case.* Six years ago a gentleman was sent to me complaining of slight but continued giddiness. I found that he showed appreciable Rombergism, that his knee-jerks could not be elicited even with reinforcement, that his pupils contracted sluggishly to light, so much so that, in one eye at least, I had difficulty in observing the reaction, and that he had been much distressed to find that in the last six months he had become impotent. At the time I had little doubt that he was suffering from early tabes, but, fortunately, I did not indicate this to the patient. He was a confirmed cigarette smoker, and in this and in other respects his mode of life had

to be altered for the better. In the course of a few months I was able to elicit his knee-jerks, and from that time onwards all the symptoms of the case gradually abated. When I saw him lately, he was, or at least appeared to be, in perfect health. Now, I am not at all certain that this patient may not in reality be an ataxic, and that he may not presently develop tabetic symptoms again. But in any case it would have been a mistake had I told him of my fears at the time of the first examination.

General rules as to mode of life.

If, however, you do not, in these early cases, indicate to the patient the exact nature of his malady, on the other hand you should not in any way make light of the matter. He ought to be warned that the condition may become a serious one, and certain general rules as to his mode of life should be laid down. That exposure to wet and cold might aggravate the malady is self-evident. Overstrain of the muscles, involving overstrain of the reflex loop, may also do much harm in hastening the breakdown of the reflex collaterals of the posterior root. I remember the case of a patient who, in the pre-ataxic stage of tabes, took one day a walk of some twelve miles. He did this to show to an

over-anxious and sceptical physician how really well and strong he was, and how absurd it was to warn him against over-fatigue. On his return from this performance, I found that he had passed suddenly into the ataxic stage.

Attention to condition of bladder.

It is necessary in these cases to make special enquiry as to the state of the functions of the bladder. If incontinence be present, you will no doubt be told of it, but if, on the contrary, there be some degree of anæsthesia with consequent very infrequent micturition, you may not be informed of this, which may indeed be complacently considered by the patient as a source of convenience and satisfaction. It is then necessary to see that the patient makes a point of emptying his bladder at regular and suitable intervals.

Dangers of sexual excess.

It is also right to caution the patient against the dangers of sexual excesses. These dangers are considerable, and the sexual hyperæsthesia, which is often a symptom of tabes, and which rises occasionally to the level of satyriasis, makes such warning the more necessary. Under these conditions the use of bromide of potassium is indicated.

A tabetic should take but little alcohol and

Alcohol and tobacco.

should not smoke, or at any rate should use tobacco only in strict moderation. His food should be plentiful and highly nutritious, and this is especially important in those cases in which gastric crises occur.

It is, unfortunately, quite clear that no measures which we can take will restore function in the degenerated neurones. The most that can be expected to occur is that we may succeed in delaying or even arresting the further extension of that process. It is to this end that the general measures used in the treatment of tabes are directed.

Anti-syphilitic treatment.

The first question which we have to decide in dealing with these cases is whether we shall prescribe a course of anti-syphilitic treatment or not. This important and difficult matter has been fully discussed already (see p. 92), and I need not repeat what has been there said.

Treatment by drugs.

Many internal remedies have been, from time to time, recommended and used in the treatment of tabes. Most of these appear to be quite useless, and of those which remain I cannot speak in any very high terms of eulogy, in so far, at any rate, as their influence on the general progress of the malady is concerned. From

time to time you will, no doubt, give tonics of various kinds, in order to improve the general health, and so, indirectly, affect the morbid process in a favourable sense. Apart, however, from such general tonic treatment, there are one or two medicines which may usefully be employed. Nitrate of silver has long been used in cases of locomotor ataxia, and I have sometimes seen benefit follow its administration. The usual dose is half a grain, given in pill form, twice a day after food. This treatment should be continued for two months. The chief disadvantage is the risk of causing argyria, but this will not occur unless the total quantity of nitrate of silver taken during the course of treatment is considerable. According to Oppenheim, anything under 10 grammes—say 150 grains—will not produce this disagreeable symptom. *Nitrate of silver.*

In iodide of potassium we possess another remedy which appears sometimes to be of considerable service. Its action is probably due, as I have previously said, to its influence on phagocytosis. It should be given in increasing doses for many weeks at a time. *Potassium iodide.*

Of the other remedies which are sometimes given with a view to arresting this disease, I

cannot give you an encouraging account. Phosphorus may be tried with some little hope of benefit. Ergot, so much given at one time, is in my opinion quite useless, and indeed harmful.

Electricity. The use of electricity in the general treatment of tabes is lauded by some, but I cannot say that I have seen much benefit follow its application. It should be employed in the shape of the constant current. One pole should be placed on the nape of the neck, the other over the lower dorsal region of the spine. With electrodes of about two inches in diameter a current from 5 to 8 m.a. is sufficient. The sitting, which should last about five minutes, ought to be repeated twice or thrice a week. The direction of the current does not appear to be a matter of moment.

Hydrotherapeutics. Hydrotherapeutic measures do not promise great things in the treatment of tabes. Nor is much direct good likely to result from a visit to Baths and Spas. So far as these expeditions are followed by improvement, this results from the beneficial effect on the general health which is produced by change of air, of scene, of diet, and of regimen.

Suspension.

In this brief survey of the general measures likely to do good in a case of tabes, there remains only one method for consideration, namely, that of suspension. This mode of treatment certainly produces, in some cases, distinct—if only temporary—amelioration of the symptoms. The original method of suspension is not to be recommended, but a very considerable stretching of the spine (the primary object of the suspension treatment) may be effected by a safer method devised by Gilles de la Tourette.[1] The patient is seated on a table, with his legs extended. His body is then pulled forward by suitably applied weights, pulleys and straps, until the face approaches the knees. He remains in this position for some minutes. According to many observers this stretching of the spine is followed in a considerable percentage of cases by distinct amelioration of symptoms. It should not be attempted in cases in which the ataxia is great, nor in those showing cachexia. The presence of arterio-sclerosis is also a contra-indication.

Certain of the symptoms of tabes demand

[1] Gilles de la Tourette, *Leçons de clinique thérapeutique sur les maladies du système nerveux*, p. 469. Paris, 1898.

Treatment of lightning pains. special treatment, either by reason of the pain and distress they occasion, or from the importance of their bearing on the general health of the patient. The lightning pains of tabes, always distressing, sometimes assume a very great severity, and they then tax to the utmost the therapeutic resources of the physician. I have already referred (see p. 302) to this matter and need not say more here, save to state briefly that you ought always to try the milder and more innocuous measures at command before you resort to the use of morphia, to which, unfortunately, you are in most cases ultimately driven. Liniments are seldom of any service, but sometimes you may find that hot baths and massage allay the severity of the paroxysm. That modified form of suspension, or rather, stretching of the spine, previously described, is often of benefit in regard to lightning pains. Faradism or high frequency currents, *Electricity.* or, better, static electricity, should certainly be employed. If these measures fail, we should then try the effect of the various anti-neuralgic remedies: salicylate of sodium, anti-*Phenacetin.* pyrine, phenacetin and the rest. Of these, so far as my experience goes, phenacetin is

the most efficient. Oppenheim recommends pyramidon highly, and I have occasionally used it with success. If these remedies do not succeed, the administration of dionine, by the mouth, may be tried. But when the pains are very severe, there is only one remedy likely to relieve the patient of his sufferings, namely, morphia administered hypodermically. Now, in a chronic disease such as tabes, in which these severe pains return in paroxysms from time to time, and in which the nervous tone and resistance of the patient tend to break down, a condition of morphinism is very liable to develop, and indeed does so frequently. No harm would be done were the administration of morphia limited to those occasions on which the paroxysms of pain are really severe. The danger consists in the use of the drug being continued in the intervals. This matter is one of vital importance to the patient and demands great tact and firmness on the part of the physician.

Dionine.

Morphia.

Its dangers.

Another symptom of a distressing and difficult nature is found in those various crises from which tabetics suffer. Here also we are usually driven, sooner or later, to the use of morphia. In cases

Gastric crises. in which gastric crises occur from time to time, it is very important to keep the nutrition high in the intervals, so that the non-retention of food during the crises may be, so far, counteracted. Leyden[1] recommends that, the moment the crisis is over, a very careful overfeeding should be instituted, on the lines of that employed, as we shall see in a later lecture, in the Weir-Mitchell treatment. *Overfeeding.* As to drugs in those cases, the use of oxalate of cerium, originally recommended by the late Sir J. Y. Simpson many years ago, has been revived. *Oxalate of cerium.* It may be usefully combined with rather large doses of bicarbonate or phosphate of sodium. In most cases of gastric crisis, however, the hypodermic use of morphia is called for. *Morphia.*

Laryngeal crises. The laryngeal crises which, if severe, produce very alarming symptoms, may be met by the inhalation of chloroform. I have seen nitrite of amyl do exceedingly well in such cases.

In the other and rarer forms of visceral crises the use of hypodermic injections of morphia is indicated.

Management of bladder troubles. The bladder disturbances, so often met with in cases of tabes, are not easy to manage. Faradism seems sometimes to do good, but the

[1] Leyden, *Die Tabes dorsalis.* Berlin, 1901.

best results I have seen were obtained by means of the galvanic current, one pole being applied over the lumbar region and the other over the perineum.

The extreme sexual irritability, to which reference has been made already, is a symptom at once very distressing and capable of unfavourably affecting the progress of the disease. This condition may be combated by the free administration of bromide of potassium. *Sexual irritability.*

The optic atrophy, which is so distressing a symptom in many cases of tabes, is, I fear, beyond the possibilities of hopeful treatment. *Optic atrophy.*

The symptom known as vertigo, is, as has been already said, the conscious and subjective expression of those pathological changes which lead to loss of the power of equilibration, and, consequently, to reeling. In the first portion of this lecture we had to consider the nervous apparatus and the mechanism by which the upright posture is maintained. It was then pointed out that various centripetal impressions combine to give that information which is necessary in order that the needful muscular adaptations *Vertigo.*

may be made—that swaying may be counteracted and the body maintained erect.

Centripetal paths for equilibration.

These centripetal paths are mainly the following:—

1. Vestibular impressions.
2. Visual impressions.
3. Muscular-sense impressions.
4. Sensations from the skin.

When speaking of the co-ordination of muscular movements it was pointed out that the necessary processes were carried out to some slight extent in the spinal cord, but, chiefly, in the cerebellum and in the cerebral cortex.

Cerebellum.

So far, however, as vertigo is concerned, we have to deal mainly with the cerebellum. For vertigo may be considered as the result on the cerebellar centres of impressions coming in from the periphere, which are contradictory and confusing. The impressions, which, when at variance, produce the sensation of vertigo, are mainly those proceeding from the semicircular canals and those from the visual apparatus. It is chiefly from these two sources that we learn the position of the head in space, and when the impressions so received are of a contradictory character, vertigo is felt. Clearly such contra-

Contradictory impressions.

diction may take various forms. Either the visual impressions may not agree with the labyrinthine, or one labyrinth may not agree with the other. All sorts of combinations are possible. But, be it produced as it may, it seems to be clear that this contradictoriness in the incoming impressions so disturbs the cerebellar centres that an impulse is sent to the sentient cortex which gives rise to the subjective sensation of vertigo. *Origin of sensation of vertigo.*

It seems, therefore, clear that vertigo, or giddiness, may result from a variety of lesions. *Causation.* It may be caused by changes in the apparatus of vision, including that presiding over the movements of the eyeballs and over the muscular-sense impressions, which tell of the tension of the ocular muscles. To this cause may be attributed the vertigo which results from diplopia, as well as that which stands in such intimate relation to nystagmus. *Visual causes.* Gerlier's disease is also very interesting in this connection.

It may, on the other hand, result from disorder of the vestibular branch of the eighth nerve, or of its peripheral apparatus. *Vestibular causes.* Under this head will be included Ménière's disease, of which vertigo is the most striking, almost the only, phenomenon. Slighter forms of vertigo may

indeed be caused by anything which tends to alter the intra-labyrinthine tension. A plug of wax in the auditory meatus may do this, or sclerotic changes in the middle ear. There are in fact many local conditions which may so act as to lead to giddiness. This auditory vertigo varies in degree, sometimes being very slight, and sometimes attaining the severity of a typical attack of Ménière's disease.

Tabetic vertigo. The vertigo seen sometimes in tabes is probably due to involvement of the neurones of the vestibular branch of the eighth nerve, or to affection of nerves connected with the ocular movements.

Central causes. So far, we have very briefly considered the more frequent of the peripheral causes of vertigo. But many and very marked conditions of giddiness result from lesion, not of the peripheral, but of the central part of this complicated but singularly beautiful mechanism. Tumours of the cerebellum are apt to produce very intense vertigo and a reeling gait. And it is exceedingly likely that the giddiness which so often attends cerebral neoplasm, whatsoever may be its site, is to be referred to increase of intra-cranial pressure affecting the cerebellum.

It is also very probable that the vertigo which results from the action of various poisons, such as alcohol, absinthe, and tobacco, from those of auto-intoxication, particularly in gastric dilatation, and from the toxines of many acute infections, may be the outcome of the action of these poisons on the cerebellar centres. It must, however, be confessed that the mode of action of many of these poisons is very obscure. We cannot, for example, say with certainty how quinine and the salicyl compounds produce the giddiness which so often follows their administration, though there is much reason to believe that they attack the vestibular nervous mechanism. Whether they act also on the cerebellar centres is doubtful. *Toxic causes.*

I need not remind you that many other lesions within the cerebrum are capable of producing vertigo. It is seen in multiple sclerosis, in cerebral hæmorrhage, in softening, in the various circulatory disturbances, in arteriosclerosis, and in many other forms of brain disease. *Cerebral causes.*

Inasmuch as the disturbances which lead to reeling give rise to stimuli which pass from the cerebellum to the sentient cortex, and seeing *Cortical causes.*

that it is only in such cortical centres that these stimuli can rise to consciousness and produce the feeling of vertigo, so it is clearly possible that disorder of these cortical centres might of itself give rise to the subjective feeling of giddiness, without there being any disturbance of the peripheral or the cerebellar mechanism. Indeed it appears to me to be likely that certain of those poisons, to which reference has already been made, which are known to produce vertigo, do so by reason of their action on the cerebral cortex. In any case we may in this fashion explain to ourselves the occurrence of that vertigo which is seen as a symptom of the neurasthenic state. It is probable that the vertiginous aura of epilepsy may also arise in a like way.

Neurasthenia and epilepsy.

Treatment of vertigo.

As regards the treatment of vertigo, it is evident that in the case of the large majority of these various causes which have been mentioned, the suitable mode of treatment is that proper to the malady of which the vertigo is a symptom. The vertigo following alcohol or tobacco is at once relieved when these poisons cease to act. When vertigo occurs in connection with epilepsy, with brain tumour, or with arterio-sclerosis, we ought to direct our treatment towards the

Causal.

primary malady. So it is with the giddiness which arises from paresis or paralysis of ocular muscles. If this can be overcome or corrected, the vertigo disappears.

Ménière's disease. We may, however, consider somewhat more fully the question of the treatment of Ménière's disease.

Causation. Aural vertigo, as has been said, may arise from various causes connected with the external or middle ear, as well as from those affecting the labyrinth. In a case of this kind, therefore, a careful search should be made for any morbid condition affecting the meatus or the tympanic cavity. In Ménière's disease the lesion is one essentially of the labyrinth itself. The predominant symptom is very intense giddiness, *Symptoms.* due to disturbance of the semicircular canals, the abnormal stimulus so created passing through the neurones of the vestibular branch of the eighth nerve to the cerebellum. Along with this there are usually signs—such as tinnitus—pointing to implication of the acoustic neurones—possibly, I am inclined to think, due to induction. To these are usually added indications of disturbance in other medullary centres, such as vomiting and vaso-motor spasm.

Treatment during attack.

The paroxysms of Ménière's disease may attain very great severity, and the suffering and prostration may be extreme. During the attack itself the patient should lie as still as possible, and the head should be steadied by means of firm pillows. In the worst cases I have encountered, what seemed to do most good was to administer a smart cathartic and large doses of potassium bromide along with stimulant. After the acuteness of the attack is over, the treatment usually adopted is that originally recommended by Charcot. Following this method sulphate of quinine is given to the amount of 10 or 15 grains daily, in divided doses. This should be continued for seven or eight days. It is necessary to warn the patient that the quinine will increase the severity of the vertigo, and indeed it is well to confine him to bed during the administration of the drug. At the end of this period the quinine is stopped, and, if success has been attained, the vertigo at once lessens. In a week the treatment is resumed and continued for a like period. These short courses of treatment may be repeated for some time. Occasionally salicylate of sodium has been used to replace the quinine, with but doubtful benefit. This quinine treat-

Bromide of potassium.

Sulphate of quinine.

ment, if it is carried on long, is said to lead to deafness. But if a patient is the subject of really severe Ménière's disease, he will certainly prefer deafness to the attacks of intense giddiness from which he suffers. I have myself seen good results from large doses of iodide of potassium in these cases, and I think this drug has served me better than quinine.

Iodide of potassium.

The counter-irritation of fly-blisters applied behind the ear is sometimes of distinct service. I am reminded of a patient who suffered from very severe attacks of this disease, and who used to beg me to order a blister even at times when I thought the counter-irritation had been sufficiently carried out. He certainly experienced great relief, and in his case the site at which the blister was most effective was just in front of the meatus.

Counter-irritation.

LECTURE IX

ON THE TREATMENT OF FUNCTIONAL NERVOUS DISEASES

NEURASTHENIA

General considerations as to functional nervous disease—Neurasthenia—Its essential features—Its heredity—Its proximate causes—Treatment of neurasthenia—Causal treatment—General treatment—Symptomatic treatment—Climatic treatment.

Functional diseases. WHEN we pass from the consideration of the organic diseases of the nervous system to that of those disorders usually called functional, we find that we have quitted the comparatively sure ground of pathological anatomy, and that we have now to deal with conditions and symptoms which rest upon no certain basis of observed microscopic change. Clinically, no doubt, the signs and symptoms of the neurasthenic condition are distinct enough when taken as a whole, though they are of an infinite variety and of an interesting complexity, tending to

pass by insensible degrees, on the one hand into conditions which are hardly removed from those of health, on the other into phases which, when more fully developed, we regard as symptoms of hysteria.

Their pathology. But, while both of these disorders—neurasthenia and hysteria—are well recognised clinical entities and are of frequent occurrence, it cannot be said that we have any true conception of the pathological changes which underlie them. That such changes do occur and are present, we cannot reasonably doubt. What their exact nature may be is a matter largely of conjecture, and we need not now pause to weigh the various hypotheses which have been advanced to explain the clinical phenomena of these interesting diseases.

Neurasthenia. Let us consider in the first place the disease known as neurasthenia. You may approximately and roughly define this malady, as indeed Beard originally did, as a state of nervous exhaustion. *Definition.* As has just been said, we do not know in this case what pathological processes have so acted on the nervous system as to produce the symptoms which we see by the bedside. We do, however, know certain things quite definitely. We

can observe that almost any set and group of neurones throughout the body may be affected, and that the physiological result is, broadly speaking, the same, whatever the point of attack may be. The essential feature in each case is that the reaction of the neurone to stimuli alters in character and in degree. You may, if you please, call this state one of "irritable weakness." Some people like this term, thinking it expresses their ideas. The phrase does not appear to me to be felicitous, nor does it illumine the subject.

Essential features.

Abnormal reaction to stimuli.

What really happens is that the nerve centres react abnormally. Stimuli which in normal people would produce but slight effect are perceived by the neurasthenic patient with much greater acuteness. For example, processes which in a normal man would produce a moderate degree of pain, may, in a neurasthenic, cause very considerable suffering. It seems indeed highly probable that stimuli which usually lie below the threshold of perception may be capable in a neurasthenic of producing an impression which rises to consciousness. This is certainly the case in regard to pathological stimuli. For example, I have seen a very slight degree of

astigmatism, so trifling that in a healthy person it would have given rise to no symptoms, produce severe headache in a neurasthenic. In the same way a quite insignificant uterine disturbance may in a neurotic woman cause a reaction altogether out of proportion to the cause. A floating kidney, a gastric disturbance,—anything which acts as a stimulus,—may in these individuals produce an exaggerated and distorted effect. It has seemed advisable to emphasise this point, since, in treating cases of neurasthenia, you will constantly meet with these exaggerated results, and it is often difficult to believe that some slight aberration from the normal condition can produce such bizarre and extraordinary effects. This consideration is of a high importance in relation to the treatment of neurasthenia.

Exaggerated responses.

A similar state of matters prevails in regard to the special senses. In connection with these, too, the response to a stimulus is, in the neurasthenic state, exaggerated and sometimes distorted. The sufferer from this malady likes to live in quietness. He cannot bear loud sounds, which indeed sometimes produce an impression rising to the level of pain. This hyperacusis is often complicated by a distressing tinnitus. Nor

Reaction to stimuli of special senses.

can he bare bright light. The stimulation of the retina produces discomfort, sometimes actual pain.

Mental response to stimuli.

In something of the same way we may explain, and so far understand, the mental phenomena displayed by a neurasthenic. His anxious fears, his deep pessimism, his morbid impulses, the temptations to wrong-doing which assail him, are all traceable to an exaggerated and morbid reaction to external impressions. They are the response to these, but, as responses, they are quite out of proportion to the external stimulus. They are exaggerated and they are distorted.

Remorse.

The remorse which these patients experience in looking back on details of their past life is very painful and is always exaggerated. A normal man ought not to entertain remorse in that way. He should certainly feel a deep and lasting regret and should truly repent of his misdeeds—and who among us is without sin? —but, having done so, he should look forward to the future and to a nobler life, and not back to the past and to his mistakes. The neurasthenic, on the other hand, is often plunged so deeply in remorse that he cannot look forward to a brighter future. His mental centres are

reacting in an exaggerated and abnormal fashion.

Giddiness. In this way also one may explain the giddiness from which such patients suffer, and those curious symptoms known as agoraphobia and claustrophobia. These are exaggerations and distortions of normal reflexes.

Ready exhaustion. Along with this condition in which the reaction to external stimuli is abnormal, there is, in neurasthenic cases, a certain weakness of neurones which leads to their ready exhaustion. In the motor sphere both of these states may be easily observed. In most of these patients the deep reflexes are exaggerated—a condition particularly well seen in connection with the knee-jerk. *Knee-jerk.* The tap on the patellar tendon which, in health, produces a contraction of the quadriceps of moderate degree, gives rise in many neurasthenics to a much more marked jerk. The response is therefore an exaggerated one. At the same time there is usually muscular weakness, and a moderate walk causes considerable fatigue.

Mental exhaustion. So it is also with regard to the neurones which subserve mental processes. These are very responsive to external stimuli, but they

are also easily exhausted. Hence come the loss of memory, the slow and laboured cerebration, and other similar symptoms in these patients.

Hereditary weakness of neurones.

Now, this peculiar state of the neurones of which I have given a few examples, and which constitutes the state known to us as neurasthenia, is due in the first instance to an inherited weakness. The patient has been born with a nervous system composed of neurones, many of which are defective in vitality, in resisting power, and in potentiality of repair. It is well known to you that certain weaknesses of tissue are very frequently met with in certain families and appear to be more or less hereditary.

Other examples of hereditary weakness of tissues.

Thus a tendency to renal inadequacy is met with in some families. In others the coats of the arteries appear doomed to early changes. More cognate to our present subject are the various hereditary or family nervous disorders, Friedreich's ataxia and the like. In these, certain groups of neurones are found to possess a low vitality and a short life. Many similar examples might be given, but enough has been said for the purpose of analogy. It is not difficult to see how, in this way, the conditions underlying neurasthenia may be looked upon as

hereditary. In different cases different groups of neurones are involved; in some the centrifugal paths, in others the centripetal, and in others still the neurones connected with the higher forms of cerebration. But in each case the neurones which have undergone change are those which, through heredity, have come to possess a less than normal vitality, lower resisting power, and less potentiality of repair. *Selective action.*

This does not, however, cover by any means the whole of the ground, nor does it explain the problem before us in its entirety. This inherited neuronic weakness is not enough to account for an attack of neurasthenia. It paves the way for the action of other causes which we may call proximate or exciting. These are manifold and varied, and we shall have to consider this matter presently in some detail, because the treatment of any individnal case of neurasthenia hinges on the question of what proximate cause is at work. In the meantime you may note on the one hand that these proximate causes will hardly produce a neurasthenia in a person who has no hereditary neuronic weakness, and on the other that that *Proximate or exciting causes.*

inherited want of vitality is not of itself sufficient to cause the disease. The onset of neurasthenia is determined by the proximate cause.

Question of diagnosis.

Before speaking of the actual treatment of neurasthenia, I may be allowed to refer for a moment to the question of diagnosis. Now, it is not, as a rule, difficult to satisfy one's self in any particular case that that disease is present. There are certain well-marked objective signs and there are many subjective symptoms which, when taken together, are amply sufficient to convince the physician that he is dealing with neurasthenia. The real difficulty which the physician has to face—and in view of treatment it is an important one—is to be quite sure that the condition is purely neurasthenia, and that there is nothing more serious behind these functional symptoms. For it must be remembered that along with the results of many organic maladies there are apt to be neurasthenic symptoms. These latter phenomena cover and mask, to a greater or a less degree, the indications of the more serious malady, and many a patient suffering from early dis-

Importance as regards treatment.

seminated sclerosis has been treated as a case of neurasthenia—a most unfortunate mistake. Therefore, before proceeding to treat any patient on the lines which are about to be laid down, you ought by minute and repeated examination to satisfy yourself that the case is certainly one of pure neurasthenia, and that the functional symptoms are not merely superadded to and masking those of some organic malady. The diagnosis, then, is one by exclusion.

Treatment of neurasthenia.

Passing now to the consideration of the treatment of the neurasthenic state in its many and varied manifestations, I may recall to your minds the importance of prophylaxis, a subject already discussed in the first of these lectures, merely adding the remark that it is in a wise and careful education that the means is to be found which may aid a predisposed individual to resist the onset of neurasthenia.

Prophylaxis.

Causal treatment.

But, when we come to consider the question of how best to treat a fully developed attack of neurasthenia, it is clearly our duty, in the first place, to discover, if we can, the proximate cause which has acted in the case with which we are dealing. And if we are so fortunate as to detect

the cause which is acting, that cause should be removed or counteracted so far as is in our power.

Various proximate causes.

It will probably conduce to lucidity if we now proceed to discuss the various proximate causes of neurasthenia and the way in which each should be specially dealt with. In considering the subject in this way, you are, however, to note that in addition to these measures suited specially to the varying causation of the condition, you will probably have to employ certain of the general measures as to diet, climate, hydrotherapeutics, and the like, which will come to be spoken of later in this lecture. These are more or less applicable to every case of neurasthenia, whatever may be its special causation.

Overstrain of neurones.

One of the most prominent and probably the most important of these causes is overstrain. The strain which acts detrimentally on those neurones which are hereditarily low in vitality may be either a mental or a bodily one. But, of these, that which operates much most frequently and injuriously is severe mental strain. Overstrain in intellectual work does not produce, as I have previously told you (see Lecture I.), serious mischief in a healthy adult, so long as the work

which he is doing is not complicated with anxiety. The reason why such work does not injure a healthy man is that it is in a sense self-limiting. After a certain period of intense intellectual labour the brain gets tired, and this fatigue is reflected in the character of the work he is turning out. He begins to perceive that the quality of his work is falling off, that it is not satisfactory, and the dissatisfaction which he naturally feels is of itself a warning to him that his brain is being overworked. He becomes irritable perhaps; most likely his sleep is interfered with, and, if he be a wise man, he stops work, goes to the country for a day or two, plays a few rounds of golf, and returns to his labour like a giant refreshed. The degree and severity of the mental work which different individuals can bear varies, as we all know, within considerable limits. Some persons can go through an immense amount of such work. I know one fortunate man who is capable of full mental vigour while obtaining only four or five hours of sleep.

Results of intense mental work.

Automatic limitation of work.

Such then is the automatic arrangement by which the brain of the healthy man, working hard but free from anxiety, limits its labour in

time to prevent catastrophe or indeed any noteworthy injury. The case stands far otherwise when material anxiety becomes added to the strain of mental work. Indeed, apart from mental work, anxiety itself produces marked exhaustion in the neurones on which that pressure falls, and this is more or less the case with most hard brain-workers. They work to make their bread, or, what is worse, to make bread for their children, and in the uncertainty which always surrounds them there is great anxiety. There comes in such cases, sooner or later, a time when the appetite fails, and when sleep becomes broken and fitful; and then there occurs the vicious circle which involves the patient in neurasthenia, because the neurones are receiving insufficient nourishment and are obtaining insufficient rest. This condition leads, in its turn, to imperfect and faulty digestion and to sleeplessness. Thus the vicious circle is completed. In this way the vitality of the neurones becomes more and more depressed, and if at the same time they are being placed under strain, then you have all the factors necessary to produce a profound neurasthenic condition. Anxiety, however, may be produced in other and less painful ways. Every under-

Addition of anxiety serious.

Vicious circle in such cases.

graduate knows the anxiety which precedes an examination, and this, combined with the hard work necessary in preparation for that trying ordeal, produces much neurasthenia. Students of medicine and students of law suffer, I think, more in this way than do those of the other faculties.

Anxiety and strain produced by examinations.

When we turn to the case of the young, the conditions are altogether reversed. A healthy child does not suffer from anxiety—indeed he hardly feels it, but, on the other hand, his nervous system may be severely damaged by over-pressure, especially at school. This is so in the case of a normal child. Still greater is the susceptibility of a neurotic child, and it is one of the unfortunate circumstances of school-life that those children who are prone to over-work their mental faculties are for the most part those who possess imagination and a high ambition, and these qualities are seen only in neurotic children. A little later, when the child is becoming a man, even the non-neurotic waken up to their responsibilities and dream of their life-work. Important examinations loom before them in the near future, and, under the stimulus of these, their work tends to become that of over-

Overstrain in children.

Over-pressure at school.

Remedy in children and adolescents.

pressure and to do serious mischief. The remedy in the case of children and adolescents is clear enough. For them it is usually sufficient to indicate a certain amount of limitation in the hours of work and a certain increase of those devoted to open-air exercise. In more severe cases it may of course be necessary to stop all mental work for a while. For the adolescent, where the damage has been great and the neurasthenia is tolerably deep, a similar stoppage of work may be necessary, but as a rule it is sufficient to limit his work somewhat, to see that that work is done rather in the morning hours than late at night, and that a reasonable amount of exercise is taken.

Limitation of work.

Exercise.

The amount and variety of exercise which a neurasthenic patient should be advised to take ought, of course, to be adjusted with some regard to his condition. Each case must be judged of on its own merits; but, speaking generally, the exercise should be of such an extent and duration as to cause appreciable fatigue. Yet that fatigue should not be so great as to prevent him from taking food with relish, on his return home. In those who are able to get about, I much prefer natural exercise

to gymnastics or to the use of such mechanical devices as the apparatus of Zander.

Choice of the variety of exercise.

The choice of the variety of exercise to be recommended is of great importance. It is essential that it should be such as may amuse and interest the patient. To tramp along an uninteresting road certainly involves exercise, but it is not likely to banish brooding thoughts, and if in this melancholy pilgrimage he is accompanied by a nurse or an attendant, that association is enough to keep his troubles before him. It is certainly not calculated to breed in him a healthy self-confidence, or to induce the feeling that he is proceeding to recovery. Indeed people have been brought to me on whom this depressing plan of treatment had been tried for weeks. They had emerged with an increased neurasthenia. What is really wanted is some pursuit of a kind to amuse the patient, and such as will force him to keep his thoughts away from his troubles. Therefore games and open-air amusements of all kinds are good. Probably, for various reasons, golf is the most suitable, but in this matter the tastes of the patient must to a large extent guide the choice.

The difficulty is greater in laying down a rule

Practical difficulties in giving advice.

of life for the neurasthenic adult whose bread depends upon his labours. In theory, no doubt, we ought to counsel him to stop work and to take a long holiday. But such counsels of perfection are hardly possible here, and it is cruel to suggest them unless the case is so severe that it is an absolute necessity that all work should cease. Short of this, one ought to advise him to vary the work during the day as often as possible, not to strain the brain too long in a given direction, to take easily digested food and as much exercise as possible.

Overstrain caused by claims of Society.

A very different variety of overstrain—the antithesis indeed of mental work—is encountered in those unfortunate persons who devote their days and nights to the claims of Society. The pursuit of pleasure is indeed the hardest work to which mankind can be subjected. It is work without rest, of constant excitement, work done in impure air at late hours, work which involves great physical fatigue, the consumption of indigestible meals, and usually a somewhat profuse indulgence in alcohol. The physical strain which is involved is increased in its morbid action by those anxieties which possess the votaries of Society—anxiety to outshine others

and to climb the social ladder. Indeed I shall leave you to fill in the sordid picture for yourselves. Much neurasthenia is produced in this struggle, and for such patients it is clear that we have to advise a return to the ordinary rules of a sane life—open air, exercise, good wholesome food; but some are so deeply involved in their troubles that nothing short of a regular Weir-Mitchell rest-cure will suffice.

Muscular overstrain.

Neurasthenia from muscular overstrain is rare. That it can occur I have no doubt, and its rarity is remarkable, seeing that without question actual organic disease of the nervous system can be produced by such muscular overstrain, where the neurones have been previously weakened. For example, I have known a spastic paraplegia follow a very long and strenuous bicycle ride in a man who was out of training.

Traumatism.

Another of the proximate causes of neurasthenia is traumatism. This particular association of cause and effect is indeed commonly enough observed. The actual injury which a patient has received plays but a small part in the production of the subsequent neurasthenia. The real cause of that condition appears to be the overpowering fear which has seized him at

Influence of fright. the moment of the accident, and therefore it is such accidents as produce a deep mental impression—earthquakes, railway collisions, and the like—which are followed by neurasthenia in those who have been exposed to them. Complicating the fright of the accident there is, in many cases at all events, an element of anxiety, for the man naturally becomes very solicitous and mistrustful as to the future and as to the possibility of his being able to resume his occupation. Probably in those who are actually injured the power of appreciating the surroundings is blunted at the moment of the accident, so that in them there is less of the element of fright. Certainly the seriously injured in a railway collision do not so frequently become neurasthenic as do those who have been less severely damaged. *Treatment of traumatic neurasthenia.* The treatment of these cases resolves itself into one of rest; they usually require Weir-Mitchell treatment, and if their anxieties as to their future maintenance are removed by large damages conferred on them by a complacent and sympathetic jury, they usually make a rapid recovery. It is only in accordance with human nature that those who are litigants in a suit in which the amount of

damages to be awarded will depend on the evidence of the severity of the injury, will not readily recover until that process of law is at an end.

In some of these cases of traumatic neurasthenia I have seen wonderfully good results follow the use of static electricity. One man who hobbled into my room with the greatest difficulty, suffering from violent pain over the lower end of the spine, the result of an accident involving severe fright, was, after a couple of weeks of such treatment, restored to apparently perfect health. *Use of static electricity.*

Fear need not be sudden in its action in order to induce neurasthenia. A slowly acting fear may be quite as effective. A patient who has a neurotic heredity may, on discovering that he is suffering from serious heart mischief, for example, pass into neurasthenia. The knowledge that he has acquired syphilis may in an even more marked way produce such fear and dread that neurasthenia results. I have seen several cases of this kind. They should be dealt with firmly. Without any attempt to ignore or minimise the importance of the venereal infection, the physician should ex- *Influence of slowly acting fear.*

plain to what an extent its results can be controlled, and how beneficial thorough treatment usually is. He should, further, strongly insist that the study of the symptoms is his duty and not that of the patient. Such cases are often extremely difficult to deal with, and require the persistent exercise of common-sense on the part of the physician.

Toxic causes. The neurasthenic state may result from the action of the toxines of the various infective diseases. Enteric fever, for example, is frequently followed by such symptoms. But probably the toxine of influenza is the most active of all these infective poisons, and many cases of neurasthenia can be traced back to attacks of that disease. These neurasthenic symptoms do not, as a rule, follow the attack of the fever in question quite immediately. Recovery takes place first, and it is only during the period of convalescence that the nervous symptoms begin to show themselves. These toxines appear in some cases—probably in most—to act as a proximate cause, producing their effect on neurones weakened by heredity, in the way I have previously explained. But in a few cases the presence of neurotic heredity

is not so conspicuous, and it appears as though the toxines produced a weakening effect on the neurones, and that the onset of neurasthenia was determined by an independent proximate cause, such as anxiety or fright. The treatment of these cases should be carried out on general lines. *Superaddition of fright.*

A profound neurasthenia may be brought about by the prolonged use of alcohol, morphia, cocaine, and other drugs. In the second of these lectures I have described the treatment appropriate to these conditions. But a more innocent indulgence may likewise produce neurasthenia, for the free and unrestrained consumption of tea or coffee is to blame for the occurrence of neurasthenia in not a few cases. Tobacco is probably rather more potent, and over-smoking, particularly if it takes the form of inhaling cigarettes, is very injurious and produces much neurasthenia in these days. On the other hand, in some cases of neurasthenia where there is much irritability, an occasional pipe may have an actually beneficial effect, and I do not think that the use of tobacco need be stopped in ordinary cases of neurasthenia, provided that there is no evidence of its having done harm, and that the indulgence be kept within narrow limits. *Alcoholic and drug causation.* *Tea and coffee.* *Tobacco.*

Digestive causes.

Among the proximate causes of the neurasthenic state, one of the most potent is disturbance of the digestive functions. Indeed it is so uncommon to meet with a case of neurasthenia in which digestion is good, that many consider the basis of the disease to rest on an auto-intoxication produced in this way. In every case of neurasthenia it is imperative to make minute inquiry into the state of the functions of digestion and the condition of the contents of the abdomen, with a view to appropriate treatment.

Nervous dyspepsia.

In not a few cases of this description it will be discovered that though there are many complaints on the part of the patient of varying subjective symptoms relating to digestion, no clear objective signs are to be found. There is no indication of gastric dilatation, or of any very distinct abnormality in the chemistry of the gastric contents, though there may be some excess or some diminution in respect of the hydrochloric acid present. In such cases you have to deal with a purely nervous dyspepsia which on the one hand is produced by the neurasthenia from which the patient is suffering, and on the other tends, by way of auto-intoxication and by mal-

nutrition, to aggravate that condition. We have here, therefore, a vicious circle, and unless the state of digestion be improved, such patients tend to sink into a deeper and deeper neurasthenia.

Treatment of nervous dyspepsia.

Cases of this kind are very difficult to treat. The patient suffers a great deal of pain, particularly after taking food, and it is natural that he should shrink from eating, and in particular that such articles of food as seem to give him most pain should be gradually omitted from the diet. As it is characteristic of this condition that pain may follow any sort of food, the list of the diet gets more and more restricted and the malnutrition and emaciation greater and greater. I am reminded of the case of a lady who under pressure of such pain had omitted article after article from her diet until, when she came to me, she was subsisting on skim-milk, to which she insisted on adding a considerable proportion of water. Her emaciation was extreme, but I assured her that in a few weeks she would be able to devour plum-pudding without trouble. This rather dangerous prediction was fulfilled in her case, but I do not think I shall venture to make it again—at least not in these precise terms.

The important thing in these cases is to get

Management of diet. the patient to take sufficient food for the needs of the body; but as nutrition increases, the powers of digestion correspondingly improve. The first step is the difficult one, and in bad cases it is sometimes necessary to resort to rectal feeding, and thus to give the stomach a few days' rest. In others a purely milk diet may be tried for a little, with the addition, after a day or two, of such artificial preparations as Benger's food or Plasmon. But, on the whole, a mixed diet is best, the foods selected being light and easily digested. It should consist largely of white meat—such as poultry, fish, or sweetbread. Vegetables, such as spinach, purée of peas, mashed potato, can usually be given with impunity; others may be tried if they are deprived of the hard vegetable fibre and carefully cooked. Eggs, either raw or very lightly boiled, usually suit well, and milk should be taken in abundance; cream also, if it can be borne. What bread is eaten should be toasted. It is important to see that the diet is varied as much as possible, and made tempting to the patient. The chief meal should be taken in the middle of the day. The patient ought not to use any alcoholic beverage, and indeed little or no liquid should be taken with

meals. The calls of thirst may be satisfied by drinking hot water about half an hour before food.

What medicines shall be employed must depend on the condition of the gastric contents during digestion. If there is hyperacidity, alkalies will in most cases afford relief, and they may be given along with such a sedative as dilute hydrocyanic acid. When the irritation is great, it may become necessary to use a little cocaine or morphia for a day or two until, by correct dieting and other means, the conditions have improved. I need not repeat how careful one should be in giving either of these drugs to neurotic subjects. In cases in which the degree of acidity of the gastric contents is diminished it is well to give twenty or thirty drops of dilute hydrochloric acid, in half a tumblerful of water, after food.

Treatment by drugs.

These indications as to diet and medicine will not, however, attain success in cases in which the nervous dyspepsia is severe and the neurasthenia deep. In such cases the Weir-Mitchell treatment is required.

Treatment in severe cases.

In patients in whom there are distinct signs of dilatation of the stomach along with the

Gastric dilatation.

symptoms of neurasthenia,—and this is, in my experience, a very common combination,—your treatment should be directed in the first instance to correct the gastric disturbance as far as may be. Here the diet requires to be very strict, much on the same lines as those already laid down, care being taken not to distend the stomach with much bulky food. In many cases lavage of the stomach gives great relief. Most usually there is some deficiency of acid in the gastric contents, and dilute hydrochloric acid should be administered. In these cases of gastric dilatation and stasis there is usually great need of gastro-intestinal antiseptics, and what I have myself found most useful is a combination of salol with grey powder or calomel, the mercurial preparation being given in small dose. I have, however, already discussed this subject.

Diet in cases of gastric dilatation.

Weir-Mitchell treatment.

But in many of these cases you will find that it is best to resort to the Weir-Mitchell treatment. This is particularly required in those patients in whom there is considerable and increasing emaciation along with weak and soft muscles.

Constipation.

The occurrence of constipation in cases of neurasthenia should be carefully avoided, especi-

ally on account of the toxic absorption which then occurs. In slighter cases the drinking of hot water night and morning may suffice to remove the constipation. The attainment of this beneficial result will be much aided by regular massage of the abdomen. But in many cases you have, at first at any rate, to resort to the use of purgatives. The best of these is, I think, a combination of aloin with extract of belladonna and extract of nux vomica. A little Carlsbad salt may be usefully added to the hot water which the patient takes before breakfast.

Hepatic and renal causes.

Just as gastric disturbance may be a potent cause of the production of neurasthenia, so you may trace the origin of the disease in other instances to disturbance of the functions of the liver or the kidney and the attendant toxæmia. The treatment of such cases should be conducted on general principles.

Reflex causes.

But, apart from toxæmia, there are certain conditions which may occur within the abdominal cavity which, apparently from reflex causes, may so act as to induce neurasthenia.

Floating kidney.

It is a matter of very frequent clinical observation to note the close association of the

eondition known as floating kidney with neurasthenia. Probably the reflex irritation whieh arises from the abnormal position of the organ is in many eases the proximate eause of the neurasthenie condition. In other cases, however, the gastrie dilatation whieh so frequently aeeompanies displacement of the right kidney aids materially in the production of the neurasthenia. That the displacement of the kidney can of itself give rise to the neurasthenie state is rendered almost eertain by the observation of eases in whieh the replaeement and retention of the kidney is followed by great improvement of the nervous symptoms. I have sueeeeded in removing the neurasthenie symptoms in quite a number of patients by controlling the movements of the kidney.

Use of a pad. In most eases the applieation of a suitable belt and pad is suffieient, but considerable eare is required in regard to adjustment, and the physieian ought to satisfy himself that the apparatus used is effective in retaining the kidney in its normal position. In bad eases, and partieularly in those in which both kidneys are freely movable and the neurasthenia is eonsiderable, the aid of the surgeon is neeessary.

Surgical measures. I have known a double nephroraphy remove, at any

rate to a large extent, the severe nervous symptoms from which the patient was suffering.

Uterine and ovarian irritation may act as the proximate cause of neurasthenia. Local treatment is to be deprecated in such cases unless there is some definite lesion which such treatment may be expected to put right in a comparatively short time. Any long-continued gynecological handling, by concentrating the mind of the patient on these organs, is apt to do harm in the way of aggravating her nervous symptoms. Such cases are best handled by the Weir-Mitchell treatment. *Uterine and ovarian causes.*

One of the most potent causes of neurasthenia is the practice of masturbation, and of other sexual vices, and a word may be said regarding these, though this subject shall be treated briefly. We have here nothing to do with the moral, or ethical, still less have we to consider the purely religious aspect of the question. We concern ourselves only with the bearing such practices have on nervous disorders, and with the lines of treatment which should be adopted. *Sexual neurasthenia.*

Masturbation, so far as its effects on the *Masturbation.*

nervous system are concerned, is probably not materially more harmful than excessive sexual indulgence of a normal kind practised with corresponding frequency. The injurious effects of this degrading habit on the character are, of course, great; but as a cause of neurasthenia and hypochondriasis its dangers lie in the frequency of repetition of the vicious act, which, in some persons, may be continued over long periods of time. The dangers generally attributed to the practice of self-abuse are certainly exaggerated. They are painted in lurid colours in the vile and pernicious publications of quacks, and the grim terrors therein described have driven many men into a profound hypochondriasis. But while I believe that there is much exaggeration in the popular conception as to the dangers of self-abuse, I am nevertheless well persuaded that these dangers are very real. That the practice of masturbation, when frequently repeated, leads to neurasthenia is quite certain, and it is not unlikely that actual organic nervous disease may take origin in this way. We know that overstrain of neurones, especially under conditions of malnutrition, is capable of causing

Its dangerous consequences.

organic changes, and probably no nervous strain could be greater than that produced by sexual excess. But while masturbation is important among the causes of the neurasthenic state, it is also one of its results. Among the signs of strongly marked neuropathic inheritance is an extraordinary precocity of sexual function, and it is not open to doubt that the instances—not so uncommon—of the practice of masturbation in infants are due to this hereditary taint. We have thus all the elements of a vicious circle. The neuropathic inheritance leads to a special liability to habits of sexual indulgence, and these in their turn tend to produce the neurasthenic state.

Sexual precocity.

Little need be said as to the treatment of cases in which the neurasthenia is either due to or increased by normal sexual indulgence carried out in excess. It is indeed but rarely that indications of the presence of such a cause will be brought to the knowledge of the physician; and most men shrink, perhaps unduly, from putting such questions as may elicit this information. If, however, the fact of such excess does come to light, then of course it is needful to enjoin moderation, remembering always that

Treatment of sexual excess

individuals differ much in these matters, and that what is moderation in one may be excess in another.

Treatment in cases of masturbation.

Turning now to the management of cases in which masturbation is being practised, clearly we must make every attempt to brace the patient's mind to determined effort towards a clean and healthy life. It appears to me to be quite essential that the physician in his dealings with his patient should carefully abstain from moral disquisitions and words of severe censure. His duty is to do his best to remove what is at once a nervous symptom and a cause of serious nervous disorder. He may leave the question of what recriminations and denunciations should be launched at the miserable subject of sexual aberration to those who consider it their business, as it is their delight, to censure morals and to brand poor frail humanity with reproach. We may surely, in this as in other matters, content ourselves with following the example of that Great Physician, who, in the presence of the adulterous woman and amid the charges of her pharisaical accusers, "with His finger wrote on the ground as though He heard them not."

As to the actual lines of treatment to be

adopted, these may be very briefly stated. The patient, having once made a clean breast in regard to this matter, should not be allowed to dwell on details, and the morbid desires which make him wish to do so should be checked. He should be fully warned of the disastrous consequences which will follow a continuance of the habit, and a strong appeal should be made to what of manliness may remain in his character. If he truly desires to throw off his vicious habit, he can do so, especially if he be aided in his strife by judicious encouragement. His life should be lived much in the open air, and he should take sufficient hard muscular exercise to produce considerable fatigue. While in the house, no minute should be left unoccupied, and by means of healthy literature, drawing, or the like, his brain should be so employed as to exclude the entry of deleterious thoughts. A sudden change of scene and surroundings is sometimes very helpful in these cases, and such foreign travel as may hold the attention by reason of the novelty of its impressions, may aid in breaking the vicious habit. Any local applications or treatment are likely to do more harm than good, and should be avoided.

Details of treatment.

General treatment of neurasthenia.

Considering now the general treatment of neurasthenia, apart from that which is specially indicated by the method of causation which may be operating in the individual case, we find one symptom which is prominent, frequent, and of great importance—namely, sleeplessness. This is perhaps more frequent of occurrence in patients in whom the disease has been induced by mental overstrain, but it may be encountered in cases of neurasthenia arising from any other cause. Insomnia is always a difficult symptom to deal with, and its results may be very serious. It is to be remembered that a neurasthenic requires more sleep than does a normal man, and if he obtains less, then a greatly increased degree of strain is placed on the weakened neurones. In dealing with sleeplessness, in such cases, the usual causes of this symptom must be eliminated. Now, I have no doubt that in the large proportion of cases of insomnia met with in practice the proximate cause of that condition is to be found in some error of digestion. Therefore when you are dealing with neurasthenic insomnia, the first thing to demand attention is the dietary of your patient. This should be carefully scrutinised, and anything which is likely to give rise to

Insomnia.

Usual causes of insomnia.

difficulty in digestion removed from the diet. It is also important, in such cases, to see that the last meal of the day is taken some little time before the patient retires to bed, so that the gastric digestion may be quiescent when the hour for sleep arrives. *Diet.*

Even in the case of a normal man it is of some consequence that deep reading and high thinking should not be maintained quite up to bedtime. If this is done, such a man may find that the activity of mental processes continues, and that some time may have to elapse before the cortex is sufficiently quiescent to allow of sleep. Much more is this the case with regard to the neurasthenic, and by him the later hours of the evening should be devoted to some light and unexciting occupation. This is a very necessary rule in cases in which insomnia is a symptom. *Working late at night.*

Various means lie to our hand for the treatment of this painful want of sleep. In some cases you may find that increasing the amount of the physical exercise your patient takes may be enough to restore sleep. The fatigue thereby produced may induce slumber, and the heightened metabolism and the increased elimination of *Treatment of insomnia.*

Exercise. excretory products which exercise promotes will aid in the attainment of that result. But it must be remembered, on the other hand, that excess of muscular fatigue, in these cases, may banish sleep; therefore one has to exercise judgment in this matter.

Warm bath. As to the other measures which are at our disposal in fighting sleeplessness, I may mention the use of a warm bath at night. Even better results may be obtained by means of a warm douche at about 90° F. continued for a few minutes. This may usefully be followed by massage. In many cases the employment of *High frequency currents.* high frequency currents has a marked effect in combating sleeplessness.

But if these and other similar measures fail of producing the desired result, and if the degree of insomnia is so considerable as to be of serious import, then we must, however reluctantly, have *Treatment by drugs.* recourse to drugs. These, if you are forced to give them, should be employed only for a day or two at a time, and the physician should take every precaution against the establishment of a drug habit. The safest of these remedies is probably bromide of potassium, and for a few nights this may be given in rather full doses. If this is

not successful, paraldehyde may be tried, and, failing that, trional.

Change of climate to remove insomnia.

For those neurasthenics who are able to move about, a change of climate may be very successful in removing insomnia. It is not possible to say exactly what climate may be of use in each case. The matter is always more or less one of experiment, and I shall have to refer presently to the question of climate in neurasthenic cases as a whole. But in the meantime it may be said as regards neurasthenic patients who are suffering from sleeplessness while living in health resorts at comparatively high altitudes, that their power of sleep returns when they proceed to climates of lower elevation.

Electrical treatment in neurasthenia.

I have incidentally mentioned the use of electricity as a means of relieving certain of the symptoms of neurasthenia which we have been discussing. We may now consider it as a remedy for the disease generally. Some observers have seen benefit follow the use of the galvanic current applied to the head and neck, but I cannot say that I have observed any remarkable improvement follow such application. Certainly, however, high frequency currents have sometimes a

beneficial effect, and I have seen remarkably favourable therapeutic results follow the use of static electricity. This is indeed the method which, above all others, is fitted for use in cases of neurasthenia.

Medical treatment of neurasthenia.

The medicinal treatment of neurasthenia is not promising. If anæmia is present, you will of course place the patient under the influence of preparations of iron. Tonic treatment may also be required, and of the various preparations at your command probably arsenic is the most valuable. In my own experience as regards the general treatment of neurasthenia, I have come to rely very much on valerian. It certainly answers well as a nervine stimulant, increasing voluntary muscular power and diminishing the sensation of fatigue. The clinical observations as to the utility of valerian are confirmed by the experimental results obtained by Féré.[1] The action of the drug appears to be chiefly on the cortex and the upper motor neurone. I have tried various preparations of valerian and have come to the conclusion that the most reliable and efficient is the ammoniated tincture. It is usual

Valerian.

[1] Féré, "Contributions a l'étude de l'action physiologique de la Valériane et des Valérianates," *Archives de Neurologie*, T. xiv. p. 22.

with me to add some tincture of asafoetida to the mixture. The taste of such a mixture is not so badly borne by these patients as one might suppose would be the case, and undoubtedly many experience comfort and are distinctly benefited by its use.

Phosphorus.

In a few cases I have found the use of phosphorus, in the form of Martindale's pill, of marked benefit. In others I have thought that the administration of thyroid extract helped me much, probably by reason of the changes in metabolism which its use produces.

Cold bath.

As a general tonic to the nervous system there are few things so good as a cold bath, and its stimulating effect is known to all of us. There are, however, few neurasthenic patients, and these examples of the malady in its lighter form, who can stand so strong a stimulus without suffering harm. In the case of this as of other forms of stimuli, the reaction, as I have so often pointed out, is, in these patients, abnormal and excessive. And therefore this, which is one of the most useful forms of treatment in the neurasthenic state, has to be modified to suit the condition of each patient. The essential point is that the

stimulus should be such as will give rise to a distinct cutaneous reaction: less than this is useless, and more than this may be very harmful. Cold bathing, in whatever form you may think right to order it, should be performed on waking in the morning.

Use in weak patients.

For those patients who are weak and in whom a reaction is easily obtained, the best procedure is that of the wet sheet. To perform this operation the attendant dips a sheet in water of a temperature of about 70° F., wrings it out and then applies it to the body of the patient from the shoulders downwards. The attendant then rubs the sheet firmly all over the body, removes it and dries the skin thoroughly with a large, rough and warm bath-towel. The temperature of the water in which the sheet is dipped should be lowered from day to day as much as the patient seems able to bear, or until it reaches about 55° F.

Wet sheet.

Douche.

A more efficient way of using cold water is that by means of the douche, and this is to be recommended for all patients who can bear it, and also for those who, at first too sensitive, have been inured to the use of cold water by means of the wet sheet. A shower of cold water at about

50° F. should be allowed to fall on the back, shoulders and limbs, and then on the chest and abdomen for a few seconds. If this is too severe a stimulus for the patient, it is well to try a douche in which the water, at first tepid or even warm, is suddenly brought down to a cold temperature. This is probably the most effective of all these methods of hydrotherapeutic treatment. In every case it is to be remembered that the object is not attained unless the patient experiences a glow of heat after the bath. Any shivering or any, even if slight, lividity shows that the means used are wrongly proportioned to the needs and sensitiveness of the case in question.

Weir-Mitchell rest cure.

I have made repeated allusion in this lecture to the method of treatment devised and recommended by Weir-Mitchell—that, namely, of which the fundamental points are rest, isolation, massage and overfeeding. This is a measure of very great value in the handling of neurasthenic patients. The details of the Weir-Mitchell method, and the manner in which I think these should be arranged for such cases, will be described in the next lecture, when we come to

speak of the treatment and management of cases of hysteria.

Climatic treatment of neurasthenia.

The last point to which I wish to refer in connection with the treatment of neurasthenia is that of the choice of a climate in those cases in which a move to the country is advisable, and is at the same time within the means of the patient. It is certainly desirable in many cases to get the patient away from those surroundings which have become associated in his mind with the various phases of his illness. Therefore, during convalescence, when a certain considerable degree of improvement has been attained, it is usually advisable to send the patient away for a time. In lighter cases, a change of climate, of scene, and of surroundings may suffice to prevent the neurasthenic condition from becoming severe, and may provide the stimulus necessary to recovery.

Travel.

I do not mean that the patient should be encouraged to make any very extensive journeyings. To move about on the Continent from town to town, filling the day with visits to churches and picture galleries, is a very exhausting process, however delightful it may be to the vigorous and healthy. For most

neurasthenics a holiday of this kind would be highly unsuitable. They require rest, as well as change of scene. Only those patients whose bodily strength is not impaired, and whose cerebral activities are such that a constant change of scene and surroundings is desirable, should be allowed to travel in this way; and even they should be warned against the risks of over-fatigue.

Conditions guiding the choice of climate.

In the case of most neurasthenic patients what one aims at is to send them to some quiet place where they can have a fair amount of sunshine, where the air is fresh and good, and where they may find sufficient interests to counteract the tedium of their stay. But before the physician proceeds to recommend a move of this sort he ought, so far as may be, to satisfy himself that the financial position of his patient is such that the expense involved will not cause anxiety. Indeed if the patient carries such cares with him, he will not benefit from any change of climate.

Choice of climate.

The choice of a suitable climate is not an easy one, but, though it is difficult to lay down any rule, it may be said generally that villages on the sea-coast are not good resorts for neu-

Seaside residence.

rasthenics. There are, of course, exceptions to this, and with some a seaside residence does well. But especially in cases in which there is irritability, excitement, gastro-intestinal trouble, or a tendency to insomnia, such resorts should be avoided.

Mountain climates.

What has appeared to me to suit best, on the whole, are mountain climates. In this country localities such as Braemar, or the upper part of the valley of the Spey are to be recommended. When it is possible to send the patient abroad, certain of the Swiss villages suit very well. The elevation should not, at first at any rate, be great; but, later on, as the patient improves in health and becomes accustomed to the climatic conditions, he may profitably move to higher altitudes, provided that he is not troubled with insomnia, and does not suffer from palpitation. I have seen excellent results in such cases, from a residence of a few weeks at an altitude of 6000 feet. The pure and rarefied air produces a singularly exhilarating effect, and provided that the patient does not over-exert himself, to which there is, no doubt, considerable temptation, the stimulus which these conditions give to the blood-forming organs and the vital processes generally,

Question of altitude.

Effect of rarefied air.

is highly beneficial. You have, however, to be careful to see that the patient does not select a village where the accommodation is bad and the food unsatisfactory. The height of absurdity would surely be reached if a neurasthenic, weighed down by digestive troubles, were to find himself in a position in which the chief viand presented for his consumption was the tough flesh of the goat.

Care as to food.

LECTURE X

ON THE TREATMENT OF FUNCTIONAL NERVOUS DISEASES—(*Continued*)

HYSTERIA

Hysteria—Its characteristics—Its causes—General rules of treatment—Causal treatment—Rules guiding the physician in relation to his patient—The Weir-Mitchell method—Isolation and other details—Hydrotherapeutic measures—Electricity—Treatment by drugs—Importance of suggestion and how it should be carried out—Treatment of symptoms—Convulsions, contracture, paralysis, headache, digestive disorders, anorexia, vomiting, constipation.

Hysteria.

If it is difficult to understand, as we have seen in the last lecture, the mechanism of production of neurasthenia, the problem is still more obscure when we try to explain to ourselves the true nature of hysteria. We know that, so far as our present power of investigation goes, there is no recognisable change of an organic nature in the nervous tissues. And yet hysteria is a definite entity, with well-marked and impressive symptoms—the stigmata of the disease

Obscurity of its pathology.

—of the true nature of which we are quite ignorant.

Mental element. We know indeed that there is a well-marked mental element governing the clinical picture. The mental disorder is not, in uncomplicated cases at least, of the nature of insanity, for those patients are usually of good, sometimes of high intelligence, and mentally clear. It is rather that the emotional centres are abnormally irritable and sensitive, and that the responses they give to external stimuli are excessive and distorted. There is also, at times at any rate, a certain enfeeblement or even loss of the power of conscious perception, which may apply to all forms of sensation or only to some.

Suggestibility. And governing all the signs and varied manifestations of hysteria there is a remarkable sensitiveness and receptivity of the higher centres which leads to easy suggestibility. Auto-suggestion is indeed, in all likelihood, the method by which many of the symptoms of this disease, such as paralysis, contracture, etc., come to pass.

Hereditary instability. In regard to the causation of hysteria, we have to recognise the influence of heredity in an even more marked degree than we did when speaking of neurasthenia. You may, I think, take

it that it is universally present, and operative in every case with which you will be called on to deal. If you cannot trace actual hysteria among the relatives or ancestors, you will probably find indications of neurotic heredity in the shape of cases of insanity, epilepsy, asthma, alcoholism and the like. It is said that the marriage of cousins is specially likely to lead to the appearance of hysteria in the offspring.

Proximate causes. Acting on this inherited instability of the cortex, many proximate causes may precipitate an attack of hysteria. Very much the same sort of cause is at work here as those we considered in relation to the etiology of neurasthenia, save that mental and emotional causes are relatively more prominent in hysteria than they are in connection with cases of nervous exhaustion. Indeed almost anything which is capable, reasonably or unreasonably, of producing a strong and distressing emotion, may, in those predisposed, be the starting-point of hysteria. Great terror, severe anxiety, deep sorrow, may all do so. Perhaps those emotional disturbances which are connected with sexual matters are the most potent. Certainly the severest cases of hysteria which I have seen have been developed as the

result of unfortunate love affairs and disastrous marriages. Certain very emotional forms of religion may likewise act as a proximate cause.

Contagiousness of hysteria.

Hysteria has a remarkable tendency to spread by imitation from one woman to another, in an epidemic form. Such epidemics swept over Europe in the Middle Ages, and they are not unknown now. This imitative hysteria is sometimes seen in a hospital ward, and not very uncommonly in schools and nunneries.

Peripheral irritation.

The proximate cause does not often take the form of a peripheral irritation. Uterine or ovarian disease may sometimes start the attack, but I fancy that it is not so much the actual local irritation which is at work, as the mental impression and emotional disturbance which the knowledge of the presence of such disease is apt to bring with it.

Alcoholism and abuse of drugs.

Chronic alcoholism, or the abuse of such drugs as morphia, may without doubt cause hysteria—at any rate in those who are predisposed.

Diagnosis of hysteria.

Before proceeding to consider the treatment of hysteria, a word may be said as to diagnosis. The necessity for an accurate diagnosis is, if possible, even greater here than is the case with

reference to neurasthenia. It is all-important that the physician should thoroughly satisfy himself that no signs pointing to organic disease are present. Unless he does this conscientiously and thoroughly, he cannot speak to the patient with that sincere confidence in the correctness of his judgment which is a *sine qua non*, if he is to be successful in treating her case. Like neurasthenia, hysteria is often present along with organic disease, the symptoms of which it masks. Your inquiries must therefore be very painstaking and thorough.

Importance of accuracy.

Prophylaxis.

We need not refer again at any length to the question of prophylaxis. This subject has been considered somewhat fully in the first of these lectures. You do not need to be reminded of the fact that these prophylactic measures are of the first importance when you are dealing with children who show signs of having inherited a tendency to hysterical manifestations.

Treatment of hysteria.

Passing now to consider the question of the treatment of hysteria, you will find that many of those measures which are useful from the point of view of prophylaxis are likewise advisable in the management of the disease itself.

General rules. A healthy and breezy, open-air life, plain but well-cooked food, early hours,—all these and similar common-sense rules already detailed are as potent in the handling of the disease itself, if you can persuade your patient to carry them out, as they are in the sphere of prophylaxis.

Causal treatment. You have next to consider whether there is any proximate cause which may be removable. As regards uterine disease, the same rule as was given when speaking of neurasthenia applies here. No local treatment which does not promise a rapidly beneficial result should be entered on. Certainly any measures which involve prolonged gynecological handling may, and probably will, do great harm.

Gynecological treatment.

Sexual aberrations. The influence of the various forms of sexual vice as a factor in the production of hysteria, and the method of dealing with these, need hardly be discussed here. I have said all that seems to be necessary as regards this subject in the last lecture when dealing with the question of the relation of these aberrations to the neurasthenic state.

The treatment of hysteria conveniently divides itself into two branches. We shall, first of all, consider those measures which are more

or less applicable to every case. Having done this we shall proceed to discuss the particular measures which are at our disposal to combat certain of the special symptoms which may arise in the hysterical state.

General measures in the treatment of hysteria.

The general measures are few in number, but of great importance. They comprise isolation, rest, massage and over-feeding—the Weir-Mitchell method. We shall also consider the use of electricity, hydrotherapeutic measures, and, perhaps more important than any, suggestion. Internal medication takes a subordinate place in the treatment of hysteria. I shall speak of these various methods presently, but before doing so I wish to refer for a moment to a matter which seems to be of surpassing importance as making for success in the treatment of hysterical patients.

Importance of the inspiring of confidence.

As determining the success or the failure of treatment in such cases, the factor which possesses the highest importance is the character of the physician himself, or, as one might say, his personal equation. On this depends the relation which he holds to the patient and the

confidence which she reposes in him. Without complete confidence and consequent obedience, all treatment is vain in these hysterical cases. The attributes on which the medical practitioner must rely in managing these difficult patients are various. Mere learning is not sufficient. A man may have graduated in medicine with the highest honours, and may be thoroughly conversant with the phenomena of hysteria, and yet be quite incapable of successfully treating a case of the kind. It is not sufficient that he is deeply learned in the science of medicine; he must also be truly a physician in the widest and broadest sense, and possess the sagacity, mental force, and general attributes which go to make up that character. It is difficult to define wherein these attributes consist, and still more difficult to cultivate them, as we all should do. But it may be said that to a thorough knowledge of the science of medicine must be added a sincere love of our Art, and a lively desire to make use of such knowledge in order to relieve or cure our patients. Together with this there must go a certain genuine interest in and sympathy with the patient by whose bedside we are seated, and a certain firm kindliness in thought and word.

Attributes of successful physician.

Importance of complete sincerity.

The physician must be true to himself. If he is so, he will also be entirely sincere in his dealings with his patient, and than this there is nothing which is more powerful in inducing confidence on the part of the patient. If, on the contrary, she perceives the slightest sign of want of sincerity on the part of the physician, all her confidence in him will be shattered, and he will not effect a cure.

Other important qualities.

The physician, to be successful in handling these hysterical cases, ought also to possess sound common-sense and tact, to have some experience of the world and of its very various inhabitants, and some degree of that quality which we know as *savoir faire.* He ought to be of quick and cultivated perceptions, capable of appraising character, of drawing rapid deductions as to the surroundings and condition of the patient.

Instinct of woman in reading character.

You should bear in mind that as you sit by the bedside examining your hysterical patient, she is also examining you. She will read your character certainly much more quickly, possibly more correctly, than you will hers. Through long generations of experience Woman has acquired for self-protection—almost as a kind of

instinct — the power of rapidly assessing the character and moral worth of Man. She must not find her physician wanting when she weighs him in these scales. By a similar instinct she reads with ease the impression her words or the phenomena of her disease are making on the physician as he examines her, and this should be carefully borne in mind. Therefore pause before you come to any definite conclusion, hear all you can, examine as thoroughly and carefully as is possible, and then make up your mind. Having once reached this point there must be no vacillation. Your opinion should be clear and definite, and your orders firm and distinct. To obtain the full effect, and to hold the confidence of your patient firm and true, you must yourself be thoroughly convinced of the wisdom of your recommendations, and you must then speak with decision and with no wavering or uncertain mind. There is indeed hardly any situation in which the practitioner is likely to be placed which is more difficult, or one which demands more self-control, sagacity, judgment and common-sense. Too much sympathy is as bad for your patient as too little, and, in examining a hysterical woman, you should be

Importance of avoiding vacillation.

Difficulties of the situation.

very careful not to let her see that you are on the outlook for any particular symptom. Such an idea would act as a powerful suggestion to her mind, and might, indeed often as a matter of fact does, give rise to the symptom in question. You are probably familiar with the mendacity and general mental perversion of certain of these hysterical patients. Their statements are to be taken with some considerable reserve, especially when they reflect on the doings of third parties.

Weir-Mitchell method.

We may now pass to the consideration of the various forms of general treatment which may be adopted in cases of hysteria. The first of these which shall claim our attention is that method introduced by Weir-Mitchell, the great value of which has stood the proof of many years of experience. It rests on four factors, more or less distinct, namely—(1) isolation, (2) rest, (3) massage, (4) overfeeding; and while certain auxiliary measures, such as the application of electricity, may be added to these, the most important factor of all, namely, suggestion on the part of the physician, is one which the isolation of the patient renders unusually potent, and which should pervade and overshadow the

Its main points.

whole of the details of the Weir-Mitchell treatment.

Isolation. Taking in the first place the subject of isolation, its value as a therapeutic agent must be apparent when you consider how useful it is even in those who are healthy. *Its value.* A man who is working his mental faculties hard, instinctively feels the need of periods of isolation. After mental fatigue there must come mental repose, and that can only be got when the man isolates himself from those stimuli which have relation to the intellectual work he has been doing. If the brain is much fatigued, the worker craves isolation, and desires to avoid, so far as is possible, all external stimuli. It is to this end that he desires to lie down in a darkened room for a little from time to time, in such depth of silence as may be attainable. No doubt the Retreats of the Religious serve a similar purpose.

Special value in hysteria. The need for isolation and the advantages which that measure brings with it, perceptible in the healthy, are much greater and more useful in those who are suffering from hysterical manifestations. This isolation cannot be carried out in the home of the patient, and if the relatives

urge the adoption of any such plan, the physician should meet their solicitations with a firm refusal.

How it should be carried out,

It is essential for many reasons that the patient should be removed from her relatives and placed in a suitable nursing establishment. Even for the sake of the relatives themselves such a separation is very desirable. A hysterical woman dominates the household which is so unfortunate as to number her among its members. Her sub-

and why.

lime selfishness governs everything and every one round her, and it is very curious, and at the same time very pitiable, to see how they believe in her and strain themselves to meet her wishes and carry out her caprices. I know a lady who for many years had served to her each day the breasts of two chickens, one at lunch and one at dinner; the legs she bestowed on her sisters, who were in reality in feebler health than she. At each meal she drank a special and very precious brand of champagne; they were served with an abominable thin claret. I speak feelingly, having had, myself, to imbibe this vile concoction. The whole house was tuned to her service. No window might be opened lest she felt a draught. No music might be played—and it was a very musical household—lest it over-excited her. The

convenience of no one within the walls of that house might be considered, save only hers. A patient of this kind, with all her absurdities and her tyranny, is a great strain on the immediate relatives. They are, however, the last to admit this, and strongly oppose any treatment which would involve removal from home and isolation. There is also another reason why, for the sake of the relatives, the patient should be removed, namely, the fear of the spread of the malady. Hysterical manifestations are, as I have said, very apt to be imitated, and for the sake of those around her it is often advisable to remove the patient, lest the contagion should spread.

Tyranny of hysterical women.

But, after all, it is the patient herself whom we ought chiefly to consider, and for her undoubtedly isolation is very beneficial. It removes her from her worries. It breaks the connection with those surroundings and conditions which have acted as stimuli, and which may even have played the rôle of the proximate cause of her malady. It gives her the favourable stimulus of fresh, new and wholesome surroundings, and brings her in close touch with healthy vigorous minds. In addition to this there is the great advantage of removing her from the sedulous and

Other reasons for isolation.

warm sympathies of her relatives. Her caprices and her various overt hysterical manifestations are fed on the effect which she sees that they have on those round her, and are indeed calculated with histrionic skill to produce the maximum effect possible. I imagine that the actors in a farce would rapidly desert the stage if all the spectators left the theatre. Let us then remove the spectators, or, in other words, let us isolate the patient.

Treatment in nursing-home,

Such isolation, to be effective, necessitates, as has been said, the removal of the patient from her home. In the case of those who are fairly well off a suitable nursing-home is the best place to carry out the treatment. It is a matter of great regret that this is so costly as to make such an arrangement impossible in the case of those whose means are straitened. For them and for the very poor there remains only the possibility of hospital treatment. But the wards of a general hospital are not so arranged as to be able to provide for the due seclusion of many patients. Still, by the use of screens placed round the bed, a fair approach to isolation can be attained, and I have seen many patients do excellently under such conditions.[1]

and in hospital.

[1] On this subject see an interesting paper by Dejerine, "Le traitement des psycho-névroses à l'hôpital par la méthode de l'isolement," *Revue Neurologique*, 1902, p. 1145.

If, as is usual in private work, the treatment is to be carried out in a nursing-home, it is necessary to select with great care the nurse who is to look after the patient. This is a very difficult matter, for out of the large number of trained nurses who are available, but a small percentage possess those qualities and that temperament which fit them for work of this kind. She must, of course, have been carefully taught the art of massage, and possess a fine touch and the needful strength and endurance. But these are not the only qualifications necessary to success. She must be *en rapport* and in full harmony with her patient. Her manners ought to be quiet and good, her character firm, her disposition bright and cheerful, and her mental endowments sufficiently high to enable her to keep her patient interested and in touch during the weeks of isolation. Indeed it is not easy to find a nurse of this kind, out of the multitude who consider themselves to be trained in massage.

Care necessary in selecting the nurse.

The second point to notice in regard to the Weir-Mitchell treatment is rest, both bodily and mental. The patient is placed in bed and kept there for the greater part of the time she is

Rest, as a factor in the Weir-Mitchell treatment.

under treatment. A due isolation could hardly be carried out under any other conditions, and for that reason alone the patient must be confined to bed. But the prolonged muscular repose is very valuable in these hysterical cases, and of equal if not of greater importance is the mental rest which the isolation involves, if it is properly carried out. In most cases it is well to keep the patient in bed for five or six weeks, and during that time she ought to make as little muscular movement as possible.

Massage. The want of exercise is counteracted by means of massage. This should be carried out vigorously by a carefully trained nurse. I prefer to have it done twice a day, beginning with a sitting of about fifteen minutes and gradually increasing the duration of the massage up to about forty minutes on each occasion. Some use the faradic current instead of the second daily massage, but I do not think the results are then so good.

Overfeeding. The overfeeding of such patients is a very important part of the treatment. It is best to *Milk.* begin by giving milk only, usually six ounces every two hours, making in all about fifty

ounces in the day. This is gradually increased until the patient is taking as much as a hundred and fifty ounces. As the massage becomes more effective, other articles of food are added, and as the diet gradually approaches a normal one the quantity of milk is diminished, though it is well to continue to administer some considerable amount, say eighty ounces daily, throughout the whole course of the treatment. The important point is to see that the patient gains in weight. The rise is usually considerable during the first week or two, after that more gradual. The temperature, which is often subnormal at the commencement of the treatment, soon comes up to a normal figure. The pulse quiets down as the days pass, and the whole aspect of the case improves. As a rule, if things do not go well, the fault lies either with the physician in that he has not succeeded in gaining the confidence of his patient, or with the nurse in that the massage is not being performed with sufficient care and skill.

Weight of patient an important means of judging of success of treatment.

Pulse.

The details of feeding and the results you may expect to obtain in favourable cases will be best understood if I give you a concrete example. I shall not trouble you with all the

notes of the case, but shall state the main facts as briefly and succinctly as I can.

Example of this mode of treatment.

This lady, whose age was about thirty, was placed under my care some years ago. She had all the marks of deep hysteria and was in a state of great emaciation, weighing only 5 st. 4 lbs. I persuaded her to go into a nursing-home, and put her on the Weir-Mitchell treatment, isolating her completely, and seeing that the massage was carefully and faithfully carried out.

Details as to food during first week.

First week.—She was at first fed exclusively on milk, 48 ounces daily; the quantity rising gradually until at the end of the week she was taking 150 ounces. On the sixth day she was given one small sandwich containing about a teaspoonful of carefully scraped raw beef. As this seemed to agree with her, she received six such sandwiches on the last day of the week. At the end of this first week her weight had risen to 6 st. 1 lb., a gain of 10 lbs.

During the second week.

Second week.—During this week the quantity of milk given varied on different days—between 135 and 150 ounces, and the administration of raw meat was continued. About the middle of the week she was given a light lunch at mid-day, consisting of chicken and farinaceous pudding,

and at the end of this week she was able to take in addition a little fish at dinner. Her weight now reached 6 st. 8 lbs., a gain, during this second week, of 7 lbs. Her temperature, which had been persistently below 96°·7, and which occasionally was more than a degree lower than that figure, came to a normal level about the middle of this second week.

During the third week.

Third week.—The patient was now able to take a substantial breakfast, lunch and dinner, and consequently the quantity of milk given was gradually reduced, until at the end of the week the daily amount was only about 112 ounces. There was again a rise in weight, though a very slight one. The patient weighed at the end of the week 6 st. 9 lbs.

During the fourth week.

Fourth week.—During this period the quantity of milk taken varied from 110 to 120 ounces. The diet was, however, made in other respects more ample. The patient was at this time taking a breakfast of egg, fish, toast and fresh butter. Her lunch consisted of mutton chop, or chicken, or rabbit, with some variety of milk pudding. For dinner she usually had either chicken or fish, followed by light pudding. At the end of this week the weight rose to 6 st. 12 lbs.

Condition at the end of treatment.

I need not trouble you with further details in this case beyond saying that at the end of eight weeks the weight of the patient had risen to within a pound of eight stone, that she was then perfectly well, and that this successful result has been maintained until now.

Hydro-therapeutic measures.

As an aid towards further efficacy in this general treatment of hysteria, you will find certain hydro-therapeutic measures very valuable. The reaction which follows the proper application of cold water to the skin is an excellent tonic to the nervous system, and cold bathing, either in the form of douches or sprays or of the wet sheet, is a valuable method of treatment in cases of hysteria. I have already described these measures in some detail when speaking of the treatment of neurasthenia.

Electrical treatment.

Electrical treatment should also be mentioned in this connection. I have said above that I consider a careful and thorough massage of the muscles is more valuable in connection with the Weir-Mitchell treatment than the method of causing contraction by means of the faradic current. This rule holds true, as I believe, in most cases; but in some the moral effect of

faradism may be useful. In these patients, a quarter of an hour or more may be spent in the evening in causing the chief groups of muscles to contract strongly, by means of the faradic current.

Static electricity. Static electricity is a valuable aid in the general treatment of hysteria. Its stimulating and tonic effect is very marked in some cases. At first it is well to use this agent in a mild way in the form of the static breeze. Later, a certain amount of sparking will provide a more powerful stimulus.

Treatment by drugs. Treatment by means of drugs is not of conspicuous service in cases of hysteria. Indeed most of these patients could no doubt be cured without the administration of any medicine at all. Still, medicines have their place in this as in other nervous disorders. Many people are sceptical as to this matter, but I am myself a firm believer in the efficacy of valerian and of asafœtida in such cases. *Valerian and asafœtida.* Reference has already been made to this subject in the last lecture, where I alluded to the observations of Féré, in which the value of valerian, well known to me from clinical experience, is experimentally established. *Bromides.* The bromides are occasionally of service

in special cases, but it is well to avoid using them without due cause. The dangers of the establishment of a morphia habit, which in hysterical cases is very pressing, should make you hesitate if you are at any time tempted to administer that drug. The suggestive effect of bread pills is lauded by some. Others, with the same end in view, administer methylene blue and expect the patient to be impressed by the corresponding coloration of the urine. I do not think you will care to use either of these remedies, or indeed to give any medicine for which there is not some reason other than the mere idea of suggesting cure. Suggestion should certainly be utilised in hysterical cases, but its employment should be a fair one and quite open and above-board. When we come to consider the management of certain of the prominent symptoms of hysteria, we shall have to refer further to certain forms of medicinal treatment which are appropriate to special conditions.

Morphia.

Methylene blue.

Use of suggestion.

We now come, in the consideration of the general treatment of hysteria, to that factor which is probably of the highest importance of all, and which ought to dominate the whole

management of the case—namely, the use of suggestion on the part of the physician. It is very difficult to describe the way in which suggestion should be used. This must, of course, vary according to the individuality of the patient, and what would act as powerful suggestion to one would be without effect and valueless on another. By suggestion, if you ask me to define it, is meant that act by which an idea is made to enter the mind of the patient in such a way as to remain there and to produce a corresponding effect. Obviously a suggestion may make for good or it may make for ill. Either of these events may happen, and both have to be taken into account; but in the first place let us consider such suggestions as have, or may be expected to have, a curative effect. No suggestion of this kind will rise to its true potency unless the physician possesses the confidence and the attention of his patient. This confidence will come, if the physician is sagacious and patient during the first few interviews. Having first satisfied himself that there is no organic lesion present, he should endeavour very quietly but insistently to induce the patient to confide the true source of her troubles to him.

Definition of suggestion.

Necessity of feeling of confidence.

This may not come at the first or even at the second time of asking, but ultimately the barriers of reserve will break down and the whole story will pour out as a flood. With overwhelmingly copious detail the patient will indicate her ideas not merely as to the proximate cause, but also as to the true nature of her symptoms. These will be ridiculously exaggerated and distorted, and it is now the moment to explain things simply but authoritatively. The physician should allow no argument. He should speak *ex cathedra*, and must not depart from that position.

Lines which should be adopted.

It is well to begin by assuring the patient that you are aware that her malady is serious to her; that you know that she suffers really and severely. This is the truth, and it is better to admit the fact frankly and fairly, instead of trying to minimise a state of matters which to the patient at least is very truly one of distress and suffering. Immediately after this admission you must, if any good is to follow your treatment, assure her that her symptoms are merely functional, that there is no organic disease, and that she will recover. This statement should be made quietly, in very clear language, and with a tone of confidence and sincerity. You

cannot, of course, make such a statement in any convincing fashion, unless you are yourself absolutely sure of its truth. A careful physical examination made previously will, however, have convinced you of the absence of any sign of organic disease.

Absurdity of patient's ideas.

After you have done your best to convince your patient of the essential curability of her malady,—and conviction will come sooner or later if you presevere in your efforts,—the next thing to do is to make her explain her ideas as to the causation of her symptoms. These will be very bizarre and absurd, and in plain and simple language you should point out how incorrect and impossible these theories are. Here again there ought to be no argument. You speak *ex cathedra.*

Argument from her present appearance and condition.

You may then perhaps point out to her that, though she has suffered so long, she does not look seriously ill; that her general condition is not what it would have been had she suffered for that period from any organic disease. If this argument is pressed home, it is usually a very efficient one. It should be followed up by repeating the firm assurance of ultimate recovery. A very powerful suggestion toward cure is made

if you ean, with perfeet sineerity, make some strongly optimistie statement to the nurse in such a way that, though the patient ean hear it, she has the impression that it was only intended for the information of the nurse.

Many other methods of curative suggestion will oeeur to you in your management of sueh cases. I need not elaborate this part of the subject further, save only to point out that one of the most diffieult and important points is to

Difficulty of inspiring patient with desire to recover.

inspire a desire to reeover. Most hysterieal patients consider recovery to be impossible, and almost all do not wish for it. They desire rather to drag on a selfish existence, and to continue to be at onee a souree of interest and a nuisance to the other members of the family. It is usually very diffieult to eonvinee the patient that sueh a view is not only foolish but also wrong. The tact of the physician ought to be suffieiently lively to tell him from what direetion the most telling argument should eome in eaeh individual case. You have to depiet in glowing eolours the joys of that kind of life whieh you have aseertained gave the patient most pleasure before her illness. You should eompassionate her for those months or years of illness during

which her life has been so sterile and uninteresting, suggesting pointedly how delightful it will be to return to such pursuits on her recovery. To patients of a high morale—and there are many such among the subjects of hysteria—the strongest appeal is that to their sense of duty and rectitude. You point, in such a case, to their duties to their relatives, so long neglected and yet now capable of being fulfilled if they will only make the necessary effort towards recovery. In many cases a strong argument will be found in the potential power of usefulness in the work of the World which the patient still possesses—a power now latent, but, if the patient makes the effort, capable of being revivified. Such are a few of the many lines of argument you may have to employ in these difficult cases.

Various arguments.

But if suggestion is a powerful instrument of good, so it may also be one for evil, under certain conditions and by reason of careless use. I have already referred briefly to this. The chief occasion on which suggestion may do harm is during the examination of the patient. In such circumstances the physician should be careful not to suggest the possible presence of a symptom. He must hear the statements of the

Harmful suggestions.

patient, but not question her as to other signs or symptoms likely to be present. The physical examination should be made minutely and carefully, but with complete reserve. Not rarely has a paralysis or a contracture dated its origin from an unguarded inquiry as to whether powerlessness or stiffness were present in a particular limb. As with motor hysterical phenomena, so with sensory; for an inquiry as to whether there is numbness in a particular skin-area will very probably cause that subjective symptom to appear in that area in which the patient believes that the physician expected to find it.[1]

Possibility of symptoms being so given rise to.

Hypnotism.

Of hypnotism as an aid in the cure of cases of hysteria I have no experience of value to give you. Indeed I am not much inclined to try it on any patient who may be under my

[1] Among the members of the Latin races hysteria occurs, probably with greater frequency, certainly with greater severity, than is the case with the population of Great Britain. Although one sees occasionally a really severe case of the disease here, it is to the hospitals of Paris, or to the description of French writers that we have to go in order to realise fully the extraordinary manifestations which may occur in this remarkable disease. It is probably in consequence of this that treatment by suggestion has, in its details, been more fully developed in Paris than has been the case in this country. For such details the works of Charcot may be consulted. I have myself derived benefit also from the perusal of a work by Camus and Pagniez, *Isolement et Psychothérapie*, Paris, 1904, in which the line of treatment adopted in the Salpêtrière is fully described, and I am glad to be able to acknowledge here my obligation to the writers of that book.

care. It is very doubtful whether it ever does good, and it is quite certain that it sometimes does serious harm.

Symptomatic treatment.

Passing from the consideration of those general measures which should be used in the treatment of hysteria, we may now turn our attention to certain means which are at our command in combating the prominent symptoms of that malady. In doing this, however, you should bear in mind that while it is right and proper to apply such special measures so as to modify the severity of these symptoms, the true treatment is one of attack on the disease as a whole, and the most useful means are those already discussed. It is useless to treat symptoms only. They may be alleviated for the moment by such measures as are about to be mentioned, but unless the hysterical condition is itself the subject of treatment, and unless the patient is brought back to a more or less normal condition, any mere alleviation of the prominent symptoms will prove of only temporary and very partial value.

The first of these special symptoms the treat-

Hysterical convulsion.

ment of which we may now consider are those convulsive attacks which, from a theatrical point of view, may be said to present the most striking feature of the malady. To those inexperienced in these matters the hysterical fit appears to be very serious, and it certainly impresses the relatives in a way and to a degree which is very pleasing to the half-conscious patient. These convulsions are no doubt dramatic, but they are not serious.

Treatment during the fit.

The patient should be placed on a mattress which has been laid on the floor, her clothing should be loosened so as to offer no impediment to free respiration and circulation, and a reasonable amount of care should be taken that in these violent convulsive movements she does not hurt herself. Any, even slight, traumatism is, however, very unlikely to happen. The guiding principle in using these various measures ought to be to let the patient feel that you are not in the slightest degree anxious as to her condition, and that you are not curious as to the phenomena of the fit, nor in any way interested in regard to the matter. If the convulsive movements are not great and the attack is comparatively mild, no special treatment should be adopted. It is

well to put screens round the mattress, to darken the room, to enjoin quiet and silence, and to let the patient feel that she is not being watched—in fact that the comedy is being played without an audience. But, if the convulsion is a severe one, certain definite measures should be adopted. There should be no attempt made to restrain the movements of the limbs by force. Any such procedure would only increase their violence. But you may find that firm pressure on the hystero-genetic points may cut the fit short. Most frequently these are to be found over the ovarian regions, in the epigastrium, or over the cardiac apex. You should therefore ascertain whether pressure made in these localities is efficacious in stopping the convulsion. If the attack continues, you should try the effect of dashing cold water on the face and of allowing the patient to inhale ammonia. If the faradic current is at hand, that stimulus, applied by means of a wire brush, may be very useful. In very severe cases it might even be advisable to administer a little ether by inhalation, though I do not remember ever to have been obliged to use this method. The physician ought on no account to be tempted to give a hypodermic

General measures.

Pressure on hystero-genetic points.

Peripheral stimulation.

injection of morphia, however severe the convulsion may be.

Importance of getting into some mental touch with the patient.

The patient, during a hysterieal convulsion, though she appears to be insensible and unconscious, is not really so. She certainly has some perception of what is going on, and is able to appreciate with satisfaction the effect which her performances have on the bystanders. She also can hear and does understand, at least to some considerable extent. Therefore, if the fit is not a trifling one and soon over, it is right that the physician should make an effort to get into some degree of mental touch with the patient, and should use what influence he may have in the way of suggestion and persuasion. There is, of course, no use in arguing with her, or in scolding her. He should speak clearly, firmly and encouragingly to her, repeating the phrases over and over, so that they may make some impression on the dulled sensorium, assuring her that the attaek will be of short duration, and that it is not of any serious import. Above all things, as has been already said, the patient must not be allowed to think that the matter is in any way an interesting one, or that she is creating a sensation. So much for the actual treatment of

the hysterical convulsion itself, but it should be remembered that the proper cure for such a state of matters is that isolation which we have just considered. It will be found, almost without exception, even in the case of patients in whom such fits have previously been of frequent occurrence, that after a day or two of isolation all such manifestations of hysteria cease.

Fits cease when isolation is carried out.

The contractures which are sometimes seen in hysteria are very resistant of treatment. When the condition is a recent one and no permanent shrinking and shortening of the muscles has had time to occur, the use of massage is likely to do good. This should be applied very carefully, and mainly to the antagonistic muscles. Passive movements of the limb should be sedulously performed and persevered in, even in spite of the pain such movements excite. The massage just referred to will be more efficacious in strengthening the antagonists if it be performed at a time when, by such passive movement, these muscles have been fully relaxed. The use of the constant current or of the faradic brush may be tried in these obstinate cases, but, so far as electricity is concerned, static treatment is a more hopeful one.

Contractures: their treatment.

Massage.

Electricity.

Isolation.

Isolation and the Weir-Mitchell treatment are usually necessary in such cases, and when the contracture is of old standing and the muscles involved are permanently shortened, the aid of the surgeon may be required. After tenotomy the antagonists, passing out of an overstretched condition, have more chance of recovering their power and successfully opposing the spasms in the agonists.

Paralysis: its treatment.

The paralysis, so often seen in hysterical cases, yields more readily to treatment, as a general rule, than does the condition of contracture of which we have been speaking. Here suggestion plays an important rôle, along with isolation, massage and passive movements, and it is usually not long before the patient can be induced to make an attempt to stand and walk. Such early attempts, in cases of paraplegia, are best made with the help of a "go-cart" in which, as the patient endeavours to walk, she can have some support under the axillæ. Faradism is of use here, and I have hastened the recovery of such a case by faradising the muscles of the legs while the patient was in the "go-cart," and during her attempts at walking.

Various measures.

Another form of paralysis which is common in cases of hysteria is that of the laryngeal muscles, causing aphonia. This hysterical aphonia is sometimes very resistant to treatment, but it often enough yields suddenly from some very slight cause. The mere attempt to examine the larynx with a mirror in the usual way—believed by the patient to be some curative measure—may be sufficient to bring this symptom to an end and to restore the voice. If this fails, it is well to try the application of a faradic current to the larynx either externally or internally; the latter, however, from its impressiveness is more likely to restore the vocal function. The external application of the electrodes is also very useful. In some cases I have found the following plan to succeed when other things failed. By this method, the two electrodes are placed one on either side of the box of the larynx, and a moderate degree of current allowed to pass. While this is going on the patient is directed to attempt to sing a note, and encouraged to make the sound more and more forcible. In this way I have seen the voice return in one sitting. *Aphonia.*

The headaches from which so many hysterical *Headache.*

patients suffer may usually be relieved by means of a eombination of eaffeine and phenacetin. The use of statie electrieity is also often of benefit. I have had very good results in many eases by using the faradic current in the following manner. The patient holds one eleetrode in her hand, you hold the other in your left hand. Moistening the fingers of your right hand you plaee them on the sealp over the seat of pain, so completing the eireuit. The current need not be strong, but should be suffieiently so to be elearly felt by the patient. The finger-tips are more sensitive than the skin of the sealp or forehead, and eonsequently you may feel with perfeet distinetness a eurrent which is not pereeptible by the patient. This is the ordinary state of matters, but sometimes the skin of the scalp in these hysterical subjeets is so hyperæsthetie that a eurrent, just pereeptible in your fingers, may produee pain in your patient.

Anæsthesia.

On the other hand, anæsthesia is common as a symptom in eases of hysteria. In addition to the general measures applieable to the hysterieal eondition, you may find that the anæsthesia ean be removed by the applieation of the faradic brush, or by statie treatment

High frequency currents may also be employed with benefit in some cases.

Digestive troubles.

The troubles connected with the digestive system, from which hysterical patients so often suffer, are many and various. These are not merely symptoms of the malady with which we are dealing, but also contribute to its causation. Their etiological relation to hysteria may, I take it, lie in one or more of three directions. The peripheral irritation produced by gastro-intestinal disorder may, in those who are predisposed, act as a proximate cause of the disease. The malnutrition which results from defective digestion may account for the onset of the hysterical manifestations, and it will certainly accentuate their severity. Finally, in the auto-intoxication which results, we may find an etiological factor of great potency.

Auto-intoxication.

These disturbances affecting the alimentary canal have a clinical character of their own. They take on the impress of the hysterical state of which they are manifestations, and they differ in important respects from the clinical expression of their similar conditions as these are seen in those who are not hysterical. These are, how-

ever, points which concern the question of diagnosis, and we need not pause to consider them here.

Importance of suggestion and isolation.

Most of the cases in which these gastro-intestinal symptoms show themselves are very difficult to treat, and, in many, a complete and prolonged Weir-Mitchell treatment will be required, during which the measures about to be mentioned may be employed. In all such patients the influence of suggestion and isolation is very great.

Spasm of œsophagus.

The spasm of the œsophagus which occurs in hysterical patients—allied probably to that very common symptom, the globus hystericus—is best treated by passing an œsophageal bougie. This is sometimes easy of accomplishment; at other times, owing to hyperæsthesia, the operation is a very painful one.

Hysterical anorexia.

The anorexia which occurs in hysterical women is a symptom which is always difficult and serious, sometimes even dangerous to life. A few cases are indeed on record in which this condition ended in death. It appears to be due to disorder of those neurones which conduct the centripetal impressions to those centres which

give rise to the feeling of appetite, or perhaps to disorder of these psychic centres themselves. We know indeed little as to the physiology of this nervous mechanism, and still less as to its pathology. But, be the mode of production of this symptom what it may, we know that patients suffering from hysterical anorexia have no desire for food—that indeed the idea of eating is repulsive to them. It is certainly wonderful how long these women can continue what is little short of an absolute fast, without showing its effects in any considerable degree. They go about, for a time, as usual and look fairly well. Sooner or later, however, the breakdown occurs. They then take to bed, emaciate rapidly, pass into a state of extreme weakness, and, as has been already said, sometimes die. The patient, regarding whom I gave you details when speaking of the minutiæ of the Weir-Mitchell treatment, was a case of this kind. The "fasting-girls" of whom one hears—the result, to a large extent at least, of the morbid taste of a section of the public which encourages such objectionable exhibitions—are probably in every case the victims of hysterical anorexia. *"Fasting girls."*

The cure of this condition is often very

Difficulty of treating hysterical anorexia.

difficult. You ought at once to insist on the carrying out of isolation and the other parts of the Weir-Mitchell treatment. Your powers of suggestion will be heavily taxed, and every means open to you of reassurance and of persuasion should be seized. At any cost the nutrition must be kept up, and, if the patient absolutely refuses to take food in any other way, you may require to have recourse to the use of the stomach tube, indeed even a nasal tube may have to be employed. The unpleasantness of these methods is itself of advantage in such cases. You have also in rectal feeding a means of checking emaciation, though the amount absorbed is not sufficient to fatten the patient.

Hysterical vomiting.

Hysterical vomiting is another symptom very difficult of management and of cure. Here also the Weir-Mitchell treatment is usually required before success can be attained. The dietary, curiously enough, appears to be of little importance. Vomiting occurs after the swallowing of any or every article of food. It is best to select such things as are at once of a high nutritive value, and at the same time not easily expelled *in toto* from the stomach. That viscus can

hardly be entirely cleared of milk, even by the most forcible vomiting. I am in the habit of giving finely-divided raw beef in these cases, for some shreds will certainly be caught and retained in the folds of the mucous membrane, when the stomach contracts, and will thus escape expulsion. The line to take is to give various articles of diet, selecting such as have a high nutritive value and are capable of very fine division. The effect, both physical and moral, of washing the stomach out is sometimes good in such cases. Rectal feeding may also aid in keeping up the nutrition. Occasionally counter-irritation does good. In one of the worst cases I have seen the vomiting ceased after I had transfixed the skin of the nape of the neck with four short acupressure needles. *Diet.*

Gastralgia, in cases of hysteria, is a symptom at once troublesome and difficult, and one which seriously interferes with nutrition. By those general measures which have been so often alluded to, this symptom can usually be made to disappear. The application of considerable heat over the region of the stomach, or the use of faradism, may aid you. As regards internal *Hysterical gastralgia.*

medication there is a strong temptation to use morphia in order to still the pain—a temptation which you should resist at all hazards. The ordinary remedies for dyspeptic cases may safely be employed. Rosenbach recommends small doses of chloral hydrate, and perhaps for a day or two this remedy may be employed without serious risk of establishing a habit.

Use of chloral.

Constipation.

Finally, a word or two may usefully be said as to the constipation which so often troubles these hysterical patients. It is not as a general rule very amenable to the action of aperients, but you usually have to give some remedies of this description in these cases until other and more general means have had time to act. A pill containing aloin with extract of nux vomica and extract of belladonna usually does well, especially if assisted in its action by the administration of an enema. But sometimes, when the constipation is very obstinate, you may have to take stronger measures in order to procure a movement of the bowels. Croton oil is sometimes required, and usually answers well. The administration of these and other similar purgatives should, however, be regarded only as a temporary measure. A better and more permanent result

Various remedies.

may be obtained by a regular and strong massage of the abdomen.

Intestinal antiseptics.

In cases of hysteria in which there are any of these symptoms from the side of the organs of digestion, I always think it well to give some form of intestinal antiseptic treatment. The danger is considerable that toxic absorption may occur from the gastro-intestinal canal, and thus an auto-intoxication may be set up. This might unquestionably tend to increase the severity of the hysterical symptoms. The methods you should employ have been previously mentioned. I have certainly seen bad hysterical cases improve when rectal irrigation was used and the tissues thus washed out.

Concluding remarks.

In bringing this course of lectures to a close, as I do here and now, it may be that I should take my leave in some felicitous and appropriate words if I could only pitch upon them. But indeed eloquent phrases are not for me, nor do I think that such are needed. I may, however, be permitted to express the earnest hope that a scheme of work which has notably helped me by systematising my ideas and focusing the results of experience, may have brought some

distinct benefit to you also,—a benefit which may be reflected on those sufferers from nervous diseases who may pass into your professional care in future years.

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